LANGUAGE DYSFUNCTIONS IN STROKE

Dissertation submitted in partial fulfillment of the requirements

for

the degree of

BRANCH – 1 D.M. (NEUROLOGY)

OF

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

DEPARTMENT OF NEUROLOGY

TIRUNELVELI MEDICAL COLLEGE,

TIRUNELVELI

AUGUST 2014

CERTIFICATE

This is to certify that this dissertation entitled **"Language dysfunctions in stroke"** submitted by **Dr. E Bobby** appearing for **D.M., Neurology** Degree examination in August 2014 is a bonafide record of work done by her under my direct guidance and supervision in partial fulfillment of regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai. I forward this to the Tamil Nadu Dr.M.G.R. Medical University, Chennai, Tamil Nadu, India.

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This is to certify that the Institutional Ethical Committee of this College unanimously approves the Thesis /Dissertation/ Research Proposal submitted before this committee by **Dr.E.BOBBY**, student of **DM (NEUROLOGY)** Department of Neurology, Tirunelveli Medical College /Hospital, Tirunelveli titled **"A STUDY OF LANGUAGE DYSFUNCTIONS IN STROKE"** registered by the IEC as 142/neuro/IEC/2011 dated. 22.03.2012. The Investigator is hereby advised to adhere to all the stipulated norms and conditions of this ethical committee.

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I, Dr.E.Bobby, solemnly declare that this dissertation **"LANGUAGE DYSFUNCTIONS IN STROKE, TIRUNELVELI MEDICAL COLLEGE HOSPITAL"** was done by me at the Department of Neurology, Tirunelveli Medical College, Tirunelveli under the guidance and supervision of the Professor of Neurology, Tirunelveli Medical College, Tirunelveli between April 2012 and November 2013.

This dissertation is submitted to the Tamil Nadu Dr.M.G.R. Medical University, Chennai – 32 in partial fulfilment of the University requirements for the award of the degree of D.M., Neurology.

Place : Tirunelveli

Date : 12.03.2014

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ACKNOWLEDGEMENT

I gratefully acknowledge and sincerely thank the **DEAN,** Tirunelveli medical college ,Tirunelveli for allowing me to do this Dissertation and utilize the Institutional facilities.

I would like to express my sincere gratitude to my Respected Professor and Head of the department, Department of Neurology, **Prof. S Saravanan, MD., D.M.,** for his motivation, guidance and encouragement in fulfilling this dissertation work.

I profoundly thank my Associate Professor **Dr.P K Murugan MD., D.M.,** for his advice, guidance and valuable criticism which enabled me to do this work effectively.

This is one another fine moment to express my gratitude and indebtedness to my Asst. Professors **DR.V Sriramakrishnan M.D., D.M., DR.M. Radha M.D., D.M., and DR.C Rachel Packiaseeli MD., D.M.,** for their supervision and guidance in making this work successfully.

I am thankful to my postgraduate colleagues for their constant support and sharp constructive criticism.

I am thankful to the Neurochemistry laboratory, Radiology Department and and all technicians who helped me to conduct this study.

I should thank each & every patient who participated in this study for their wholehearted cooperation in making this study successfully.

CONTENTS

INTRODUCTION

Worldwide stroke is one of the leading causes of death, with stroke mortality being very high in Asia and Eastern Europe according to WHO (2004). The annual stroke death in developing countries by 2020 is estimated to be around 19 out of 25 millions (1). Stroke not only causes death, but also worse disabilities in adults.

Among the stroke survivors, approximately 30% require assistance for daily living, 20% require assistance for ambulation and 16% require institutional services.

Among the disabilities of stroke, post stroke language disorders are frequent which include aphasia, alexia, agraphia, acalculia and apraxias (2). Aphasia, being a common consequence of left hemispheric lesion is one of the most common neuro psychological consequences of stroke, prevalent in nearly one third of all stroke patients. Recovery pattern of aphasias vary in various aphasias.

Aphasia, the loss or impairment of language caused by brain damage, is one of the most devastating cognitive impairments of stroke. Aphasia is observed with the frequency ranging from 21% to 38% in acute patients, and stroke accounts for most of the new cases of aphasia seen in neurological practice. The community incidences 43 / 1,00,000 per year and the prevalence is 3000 per million (3). The presence of aphasia is an index of poor prognosis, with more severe motor,

cognitive and social disability (4,5) and higher mortality (5,6). Aphasia outcome remains poor – 32% to 50% of aphasics still suffer from aphasia six months after stroke. Therefore it is important to evaluate and treat post stroke aphasias.

The incidence of stroke follows a rising trend in India. Post stroke language disorders are frequent and disable the quality of life. There is necessity to differentiate and classify aphasias for prognosis and outcome assessment.By more precise and detailed description of the language functions of the brain damaged subjects, it is possible to plan novel reeducative strageties and to monitor their recovery in performance. Early rehabilitation for language dysfunction and other associated neurological deficits restores the quality of life in affected subjects. Aphasia treatment have so far have been given less importance by physicians and speech therapists. Studies regarding language dysfunction are less in our place.

The aim of this study is to determine the characteristics of aphasia in stroke, the influence of age, sex and various other risk factors, the type of aphasia, its severity and to correlate the type of aphasia with imaging studies.

AIM OF THE STUDY

The aims of the study are

1. To find out the different types of aphasias prevalent among adult stroke patients attending Medicine and Neuromedicine departments of Tirunelveli Medical College hospital.
2. To study the recovery patterns among the different types of aphasias.
3. To correlate the types of aphasias with the imaging CT/MRI.
4. To study the factors influencing the outcome of Aphasias.

MATERIALS AND METHODS

Study Design:

This study is a single centered hospital based,cross-sectional,descriptive study.

Study Population:

Study was carried out in patients attending medicine and Neuromedicine departments of Tirunelveli medical college hospital with stroke and language dysfunctions.

Period of study:

The study was conducted during the period of April 2012-November-2013.

Ethical Approval:

Study was approved by the Institutional Ethical Committee.

Consent:

An informed written consent in Tamil was obtained either from the patients or relatives included in the study(Appendix 8).

Sample Size:

A total of 100 stroke patients with language dysfunction.

Inclusion Criteria:

Adult stroke patients of age 18 to 80 years with language dysfunction, attending medicine and Neuromedicine departments (both outpatients and inpatients) , with confirmation by CT/MRI and willing to participate in the study were included.

Exclusion Criteria:

Patients with

1. Psychiatric disorders
2. Developmental dyslexia
3. Head injury, space occupying lesions etc
4. Stuttering speech
5. Disorders of phonation
6. Demented patients
7. Mental retardation
8. Morbid illness like end stage renal disease, cardiac failure, corpulmonale
9. Patients not willing to participate

METHODS OF STUDY

In all patients epidemiological data including age, sex, literacy status and presence of risk factors for stroke like hypertension, diabetes, heart disease, smoking, alcoholism, family history were collected. Patients handedness was assessed using handedness questionnaire either from the patient or care givers(Appendix 8).

General physical examination and neurological examination was done in all patients. Routine biochemical investigations were done. Neurological status was assessed using National Institutes of Health Stroke Scale. It contains 11 items with the minimum score 0 and the maximum score 42. The reliability (7) and validity (8) of the NIHSS is well documented. (Appendix (8)

NIHSS-0 – Normal examination

NIHSS 1-7 – Mild neurological deficit

NIHSS 8-14 – Moderate neurological deficit

NIHSS > 15 – Severe neurological deficit

ASSESSMENT OF LANGUAGE FUNCTION

Bedside assessment of language was done in all patients as follows

1. Spontaneous speech

a. Informal interview
b. Structured task
c. Automatic sequences

2. Auditory comprehension
3. Naming
4. Repetition

The first step is to listen to spontaneous speech. The patient is asked simple questions like describing the weather outside or the reason for consulting the doctor. The following observations are done from the patient's speech like initiation difficulty, fluency, articulation, phonation, speech rate, prosody, length of the phrase, word finding difficulties, presence of circumlocution, paraphasias and neologisms.

Fluency was assessed using animal naming test in one minute. Normal individuals can name 18-22 animals in one minute. A score less than 13 suggest impairment in verbal fluency.

Naming ability was tested with confrontation naming test. A variety of objects or pictures are pointed to and the patient was asked to name them. Several categories of objects were used (colours, body parts, articles of clothing and parts of objects).

Comprehension was tested by asking patients specific questions that can be answered with a “yes” or “no” response or by asking patients to point out various objects in the room.

Repetition of spoken language was tested by presenting material in ascending order of difficulty beginning with single monosyllabic words and proceeding to complex sentences. Normal people can usually repeat sentences of 19 syllables or 6 words accurately.

In patients who were found to be aphasic by clinical language examination, detailed language function was done based on western aphasia battery (kertesz, 1979, 1982 (9))(Appendix 8). The battery provides insight into patients speech and language function and groups patient into various aphasia syndromes. The WAB has four subtests – spontaneous speech (fluency and information content), comprehension, repetition and naming. The spontaneous speech subtest carries a maximum score of 20 while other three subtests carries a maximum of 10 each.

The Aphasia Quotient is computed using the subtest scores (the sum of all the subtest scores multiplied by two). Language is classified as normal if AQ of 93.8 or more is achieved. Patient’s sub scores on fluency, comprehension, repetition and naming permit classification of language impairment according to the taxonomic

table. These discrete cut off scores in the WAB criteria for classification are based on Kertesz's review of 150 patients.

The questionnaire was translated in tamil, which is the native language in our place. The battery was administered thrice, during first admission in hospital, 12 weeks and 24 weeks.

Classification of Aphasias

Aphasics are subdivided into groups based on the initial scores in the following table 1. Most important factor is fluency which divides the aphasics into nonfluent global, Broca, transcortical motor aphasias from fluent Wernicke's, conduction and transcortical sensory aphasias. Next is the comprehension which seperates Broca from global, Wernicke from anomic aphasia, conduction aphasia and the third is repetition differentiating transcortical aphasics and conduction aphasias from other varieties of aphasia.

Table 1

Criteria for classification of aphasia

Types of Aphasia	Fluency	Comprehension	Repetition	Naming
1. Global	0-4	0-3.9	0-4.9	0-6
2. Broca	0-4	4-10	0-7.9	0-8
3. Wernicke	5-10	0-6.9	0-7.9	0-7
4. Conduction	5-10	7-10	0-6.9	0-9
5. Anomic	5-10	7-10	7-10	0-9
6. Transcortical Motor	0-4	4-10	8-10	0-8
7. Transcortical Sensory	5-10	0-6.9	8-10	0-9

Identification of cortical language areas in CT/MRI scan

CT scan brain was taken in all patients and MRI in affordable patients. The corresponding CT slices were labeled sequentially from the base towards the vertex according to known cortical language areas present in each slice. Broca, Wernicke, Supramarginal and Angular gyrus areas have had easily identifiable relationships to the specific parts of the ventricular system as discussed below.

1. **Broca's cortical area**

Situated in the frontal lobe, lateral to the inferior portion of anterior horn of the left lateral ventricle. Cortical representation of the area is present lateral to the anterior horn of the left lateral ventricle.

2. **Wernicke's cortical area**

This area (area 22) is present, lateral to the third ventricle and the quadrigeminal cistern in the temporal lobe. The area is lateral and just anterior to the atrium of the left lateral ventricle. The density of the calcified choroid plexus often present in the atrium is a useful anatomic landmark relative to the Wernicke's area on CT scan.

3. **Supra marginal and angular gyrus areas**

The supra marginal and angular gyrus cortical areas (areas 40 and 39) are usually observed, lateral to the posterior portions of the body of the left lateral ventricle.

Lesions whether infarct, haemorrhage, haemorrhagic infarct, cortical, sub-cortical or combined were also taken into consideration. The size of the infarct was calculated from the maximum horizontal and vertical diameter in the CT and the volume of the haemorrhage was calculated using the formula abc/2

a- maximum transverse diameter

b- maximum vertical diameter

c- no of slices of CT with bleed

Statistical Tools

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using SPSS – version19 statistical package.

Using this software range, frequencies, percentages, means, standard deviations, ‘t’ value, chi square and 'p' values were calculated. Student’s ‘t’ test and ANOVA test were used to test the significance of difference between quantitative variables and Yate’s or Fisher’s test for qualitative variables. A 'p' value less than 0.05 is taken to denote significant relationship.

REVIEW OF LITERATURE

Speech and language functions are of fundamental human, significant both in social interaction and in private intellectual life. In general “language” should be separated from “speech” which refers to the neuromechanical process of articulation.

Language is a complex system of communication symbols and rules for their use. Speech is the articulation and phonation of language sounds.

Aphasia is an acquired language disorder causing deficits of production and comprehension of verbal messages in individuals with a normal language acquisition history (10).Aphasia can involve the entire linguistic system, but can also impair single components or modalities: phonology, lexicon, morpho-syntax and semantics. Input and output, oral and written language

Aphasia does not include

1. Developmental disorders of language, often called dysphasia;
2. Purely motor speech disorders, limited to articulation of speech, referred to as stuttering, dysarthria, and apraxia of speech; or
3. Disorders of language that are secondary to primary thought disorders, such as schizophrenia.

Encompassed under the term aphasia are selective, acquired disorders of reading (alexia) or writing (agraphia).

Elementary review of linguistic components

1. Phonemes - the smallest meaning carrying sounds (11);
2. Morphemes - the smallest meaningful units of a word (11),
3. Morphology - use of appropriate word endings and connector words for tenses, possessives, and singular versus plural;
4. Semantics/Graphemes refers to word meanings.
5. Lexicon is the internal dictionary.
6. Syntax is the grammatical construction of phrases and sentences.
7. Discourse refers to the use of these elements to create organized and logical expression of thoughts.
8. Pragmatics refers to the proper use of speech and language in a conversational setting, including pausing while others are speaking, taking turns properly, and responding to questions.

Approximately 90% of people are right handed 10% are non right handed (left handed and ambidextrous). The planum temporal on postero superior surface

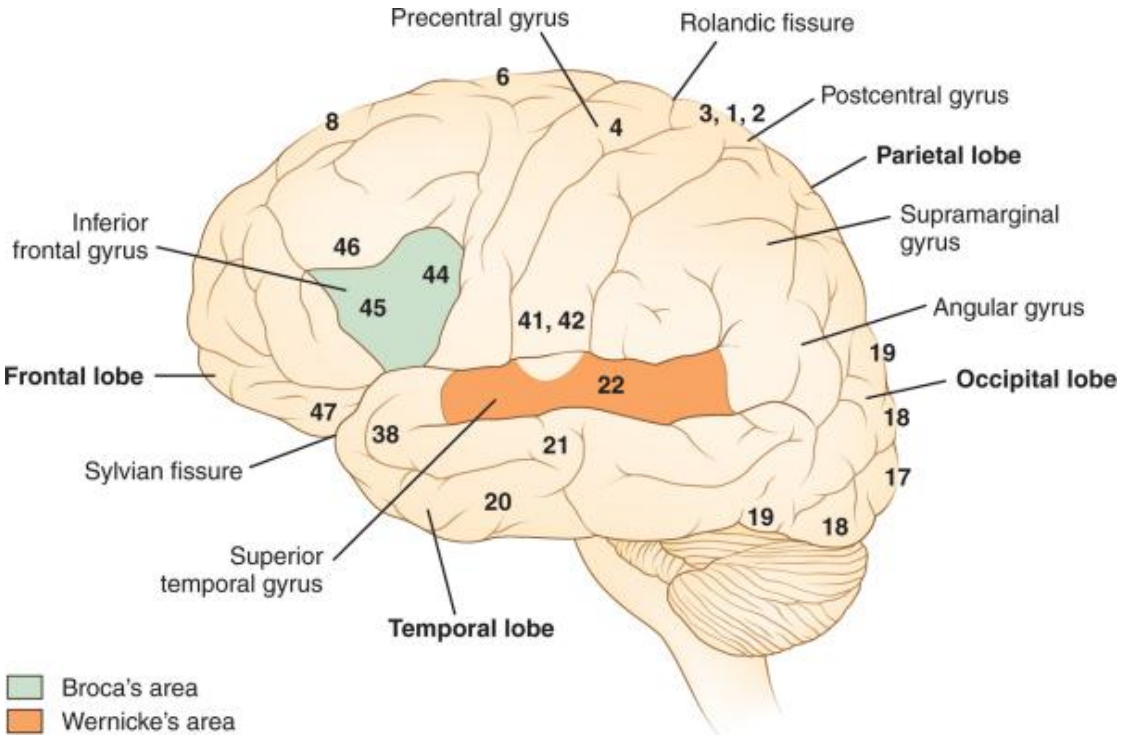
of temporal lobe is larger on left side in about 2/3 of population. The left hemisphere controls speech in 98% of dextral and also in about 50% of the non right handed persons explaining low incidents (2% - 3%) of crossed aphasia (12).

HISTORICAL BACKGROUND

Modern study of aphasia began with the publication by Paul Broca (1861) of a postmortem study on a patient with speech difficulty (13). Broca attributed this aphasia to the lesion of the posterior portion of inferior frontal gyrus, now called Broca's area. Later Broca brought to the world the fact that left hemisphere was dominant for language.

Carl Wernicke (14) (1874) published 'Der Aphasische Symptometon Complex' in which he explained the disorder of comprehension with fluent form of aphasia and associated the lesion in the posterior portion of superior temporal gyrus, now called Wernicke's area. Wernicke's paper paved way for further research in aphasias.

Anatomy of language



The main language areas are situated in most persons, in the left cerebral hemisphere. The entire language zone lies around the perisylvian region i.e it borders the sylvian fissure. Two language areas are receptive and two are executive, concerned with the production of language. The main language areas are Wernicke's area (area 22) at postero superior temporal region, Broca's area (area 44, 45) at the posterior end or inferior frontal convolution, angular gyrus (area 39) in the inferior parietal lobule, the supramarginal gyrus lying between the auditory

and visual language centers just anterior to the visual association cortex, Exner's writing area in the posterior part of the second frontal convolution.

The Wernicke's area is concerned with the perception of spoken language along with the areas 41 and 42 (Heschl's gyri). Perception of written language carried out by the angular gyrus, anterior to the visual receptive areas. Supramarginal gyrus integrates the visual and auditory language functions. Broca's area mediates the motor aspects of speech. Exner's writing area concerned with the writing act.

These sensory and motor areas are connected by rich network of nerve fibres, arculate fasciculus and subcortical white matter of the insula. Many additional corticocortical connections leading to the perisylvian zones and project from them to other parts of the brain. The visual receptive and somatosensory zones are integrated in the parietal lobe, and the auditory receptive zones in the temporal lobe. Short association fibres join the Broca's area with lower rolandic cortex, innervating the muscles of lips, tongue, larynx and pharynx. Similarly the Exner writing area is integrated with the motor apparatus for the muscles of the hand.

The perisylvian language areas are also connected with the striatum and thalamus and with corresponding areas in the non dominant cerebral hemisphere

through corpus callosum and anterior commissure.Damage to input or output modalities of areas of central language process results in aphasias (1).

CLASSIFICATION OF APHASIA

Aphasia can be broadly divided as fluent and non fluent (Benson 1967) or as anterior and posterior (Goodglass and Kaplan, 1972), or as a motor and sensory (originally proposed by Wernicke). The Boston Aphasia Classification System, classifies the aphasia syndromes into eight types (15) .

1. Broca's aphasia
2. Wernicke's aphasia
3. Conduction aphasia
4. Global aphasia
5. Transcortical motor aphasia
6. Transcortical sensory aphasia
7. Isolation aphasia
8. Anomic aphasia

Broca's Aphasia

In Broca's Aphasia the pattern of speech is nonfluent with the production of 10 to 15 words per minute. The patient speaks with hesitation, producing the principal, meaning-containing nouns and verbs with omission of morphemes and

small grammatical words. This is termed as telegraphic speech or agrammatism. Wide range of variation in the motor speech deficit occur. The mildest poverty of speech and so-called cortical dysarthria with entirely intact comprehension and ability to write (Broca's area aphasia;"mini-Broca"). The complete loss of all means of lingual, phonetic, and gestural communication.

Paraphasia is a speech error in which the patient substitutes a wrong sound or word for the intended sound or word. Paraphasic errors may be phonemic(literal) where there is addition ,deletion or substitution of phoneme or semantic, where the patient substitutes a wrong word. Phonemic paraphasias are characteristics of anterior lesions and semantic paraphasias in posterior perisylvian lesions .

Paraphasic errors in naming more frequently are of literal type; they make many phonemic errors . Naming difficulty is present. Patients may repeat a few stereotyped utterances over and over again, as if compelled to do so—a disorder referred to as monophasia (Critchley), recurring utterance (Hughlings Jackson), verbal stereotypy, or verbal automatism.

The Auditory comprehension is intact. On detailed examination complex comprehension is impaired. Repetition is impaired. Reading syntax is difficult.

Writing is also difficult. Letters are malformed and words misspelled. While writing to dictation is impossible, letters and words can still be copied. The patient may be able to sing inspite of dysarthria.

The patient recognizes his mistakes. Repeated failures in speech cause exasperation or despair. They very frequently develop depression (16).

"Pure" Word Mutism (Aphemia, Pure Motor Aphasia Of Déjerine)

It is a variant of Broca's aphasia. The patient initially is mute and starts to speak with substitution of phonemes and pauses, with other language functions being normal. This is due to small lesions in Broca's area and its adjacent subcortical white matter or of the inferior precentral gyrus.

Wernicke's Aphasia

The expression of speech is fluent with impaired comprehension. Speech contains large numbers of function words (e.g., prepositions, conjunctions) but few substantive nouns or verbs that refer to specific actions. The output is therefore voluminous (logorrhea) but uninformative.

The speech is empty without meaning, filled with neologisms (formation of new words), verbal paraphasias and jargon speech.Frequent paraphasias and neologisms, combined with agramatism and high word output result in completely

unintelligible speech, termed Jargon aphasia or word salad.Gestures and pantomime do not improve communication.

Patient has difficulty in naming with impaired repetition, reading and writing.

The deficit associated with Wernicke's aphasia may be a partial or complete right homonymous hemianopia. As the patient is unaware of his problem, the chances of depression are less.

Pure Word Deafness

Rare but striking syndrome of isolated loss of auditory comprehension and repetition, without any abnormality of speech, naming, reading, or writing. The lesion is usually in the bilateral or left superior temporal gyrus which affects the flow of information from the unimodal to Wernicke's area.

It is a disconnection syndrome due to the loss of white matter connections.. In time, patients with pure word deafness teach themselves lip reading and may appear to have improved.

Global Aphasia

Global aphasia is a combination of Broca's and Wernicke's aphasia. The speech of the patient is nonfluent or mute, with poor comprehension, repetition, naming,

reading, and writing. Associated signs in the patients are right hemiparesis, hemisensory impairment, and hemianopia.

The patients have large lesions with the involvement of inferior frontal, superior temporal regions, and parietal lobe. Patients with the sparing of superior temporal gyrus have tendency to recover their auditory comprehension and they evolve into Broca's aphasia. The recovery is prolonged in global aphasia.

Conduction Aphasia (Central aphasia/ Deep aphasia)

Speech output is fluent, comprehension of spoken language is intact, and repetition is severely impaired .Patient may have literal paraphasias. There may be naming difficulty with intact auditory comprehension. Repetition is worst affected.

Reading aloud is impaired, but reading comprehension is preserved. Wernicke's aphasia may evolve into Conduction aphasia. It is one of the disconnection syndrome. The lesion is usually present in arcuate fasciculus.Benson et al suggested that patients with limb apraxia have parietal lesions, temporal lesions in those without apraxia (17). Lesions of supramarginal gyrus or temporal lobe may also be present supramarginal gyrus is responsible for auditory immediate memory and phoneme generation and lesions of this area results in conduction aphasia and phonemic errors (18).

Anomic (Amnesic, Nominal) Aphasia

Naming difficulty is the principal deficit. There are typical pauses in speech, groping for words, circumlocution, and substitution of another word or phrase that is intended to convey the meaning. Articulation, comprehension, and repetition are intact, but confrontation naming, word finding, and spelling are impaired. Inability to produce nouns is characteristic of temporal lobe lesions whereas, inability to produce verbs occur in frontal lesions (19).

Anomic aphasia is the single most common language disturbance seen in head trauma, metabolic encephalopathy, and Alzheimer's disease. No specific areas could be localized for this aphasia.

Transcortical Aphasia

Analogs of the syndromes of global, Broca's, and Wernicke's aphasias, with intact repetition.In transcortical aphasias the causative lesions do not disrupt the perisylvian language circuit from Wernicke's area through the arcuate fasciculus to Broca's area. Instead, these lesions disrupt connections from other cortical centers into the language circuit—hence the name transcortical. An analog of Broca's aphasia in which speech is telegraphic, comprehension is relatively spared, but repetition is fluent.

This syndrome occurs with lesions in the frontal lobe, in the deep frontal white matter, or in the medial frontal region, in the vicinity of the SMA. All of these lesion sites are within the territory of the anterior cerebral artery, separating this syndrome from the aphasia syndromes of the middle cerebral artery (Broca's, Wernicke's, global, and conduction aphasia). An analog of Wernicke's aphasia in which fluent, paraphasic speech, paraphasic naming, impaired comprehension, and abnormal writing coexist with normal repetition. This syndrome is relatively uncommon, occurring with strokes of the left temporo-occipital area and in dementias.

Mixed transcortical aphasia (Isolation Syndrome)

An analog of global aphasia in which the patient repeats, but has no propositional speech or comprehension. The patient may parrot fragments of heard conversations (echolalia). The lesions present as large, watershed infarcts of left hemisphere. Lesions are patchy and can be associated with anoxia, carbon monoxide poisoning.

Subcortical Aphasias

Two types of subcortical aphasias are described.

- Thalamic lesions
- lesions of the subcortical white matter and basal ganglia.

Thalamic Aphasia

Left thalamic lesions present with the Wernicke's -like fluent aphasia, but the comprehension is better when compared with the cortical Wernicke's aphasia. Reading and writing may or may not be affected.

Characteristically, the patient's ability to repeat dictated words and phrases preserved.

The patient fluctuates between an alert state and drowsy state. The language is near normal in an alert state with paraphasias and poor comprehension in a drowsy. can occur even with a in a Left-handed patients with right thalamic lesion may present with thalamic aphasia indicating the hemispheric language dominance of thalamus.

Aphasia in Basal Ganglia

Lesions of the anterior caudate, putamen and deep WM commonly produce Broca's-like aphasia. Patients with this lesion have an "anterior subcortical aphasia syndrome" involving dysarthria, decreased fluency, mildly impaired repetition, and mild comprehension disturbance. This syndrome most closely resembles Broca's aphasia, but with greater dysarthria and less language dysfunction More posterior lesions involving the putamen and deep temporal WM, are associated with fluent, paraphasic speech and impaired comprehension resembling the features of

Wernicke's aphasia. Finally, larger subcortical lesions involving both the anterior and posterior lesion sites produce global aphasia.

Alexia(Word blindness, text blindness, visual aphasia)

Acquired sensory aphasia, inability to read in a previously literate person.

It may present as

1. Alexia with agraphia
2. Alexia without agraphia
3. Alexia with aphasia(Frontal alexia)

Neurolinguistic model of the reading process. Evidence from studies of the alexias points to three separate routes to reading: 1, the phonological (or grapheme-phoneme conversion) route; 2, the semantic (or lexical-semantic-phonological) route; and 3, the nonlexical phonological route.In deep dyslexia, only route 2 can operate; In phonological dyslexia, 3 is the principal pathway; In surface dyslexia, only 1 is functional.

Aphasic Alexia

Alexias are divided into four patterns based on the breakdown in specific stages of the reading process : letter-by-letter, deep, phonological, and surface dyslexia

Letter-by-letter dyslexia is equivalent to pure alexia without agraphia. In Deep dyslexia patients is able to recognize and read aloud familiar words with semantic or visual errors in reading and not able to read nonsense syllables or nonwords. Word reading is not affected by word length , for example, one could read “ambulance” but not “am.”

In Phonological dyslexia, similar to deep dyslexia, patient is not able to read nonwords, but able to read single nouns and verbs. Patients appear to read words without understanding. Surface dyslexia involves spared ability to read laboriously by grapheme-phoneme conversion but inability to recognize words at a glance. These patients can read nonsense syllables but not words of irregular spelling, such as “colonel” or “yacht.”

"Pure" Word-Blindness (Alexia without Agraphia, Visual Verbal Agnosia)

Visual Analogue of Pure word deafness. Writing is preserved with the inability to read what is written. Literate person loses the ability to read aloud, to understand written script, and, often, to name colors, to match a seen color to its spoken name—visual verbal color anomia. Understanding spoken language, repetition of what is heard, writing spontaneously and to dictation, and conversation are all intact.

Associated deficits include right hemianopia and frequently deficit of short-term memory. Lesion involves the left visual cortex and geniculocalcarine tract, as well as the callosal connections of the right visual cortex (Disconnection Syndrome).

Alexia with Agraphia

It is the inability to read or write in a previously literate person. Usually speech, naming, auditory comprehension and repetition are intact. Most of the patient have a fluent speech with paraphasias and naming difficulty.

Associated deficits include right hemianopia and elements of Gerstmann's syndrome: agraphia, acalculia, right-left disorientation, and finger agnosia. The lesions typically involve the inferior parietal lobule, especially the angular gyrus. Etiologic disorders include strokes in the territory of the angular branch of the left MCA and mass lesions in the same region.

Agraphia

Writing may be affected either in isolation (pure agraphia) or in association with aphasia (aphasic agraphia). Isolated agraphia has been described with left frontal or parietal lesions.Agraphias can be analyzed in the same way as for the alexias

Phonological agraphia involves the inability to convert phonemes into graphemes, or to write pronounceable nonsense syllables, in the presence of ability to write familiar words. Deep dysgraphia is similar to phonological agraphia, but the patient can write nouns and verbs better than articles, prepositions, adjectives, and adverbs. Lexical or surface dysgraphia, patients can write regularly spelled words and pronounceable nonsense words but not irregularly spelled words.

Language In Right Hemisphere Disorders

Left-handed patients may have right hemisphere language dominance and may acquire aphasic syndromes. Right-handed patients occasionally become aphasic after right hemisphere strokes, a phenomenon called crossed aphasia rare, occurring in only 1 percent of cases (Bakar et al., 1996). These patients presumably have crossed or mixed dominance. Even right-handed persons with typical left hemisphere dominance have altered language function . Affective aspects of language are impaired, such that the speech sounds flat and unemotional; the normal prosody, or emotional intonation, of speech is lost (Aprosodias). Motor aprosodia involves loss of expressive emotion with preservation of emotional comprehension;

Sensory aprosodia involves loss of comprehension of affective language, also called affective agnosia. These deficits significantly impair patients in the

pragmatics of communication. In other words, right hemisphere–damaged patients understand what is said, but not how it is said. The related issue of the accent of speech, which carries such a strong regional identity, probably also has an anatomic meaning.

Recovery And Rehabilitation Of The Patient With Aphasia

Patients with aphasia from acute disorders, such as stroke, generally show spontaneous improvement over days, weeks, and months. The greatest recovery occurs during the first 3 months, but improvement may continue over a prolonged period. Aphasia type changes during recovery: Global aphasia evolves into Broca's aphasia, and Wernicke's aphasia into conduction or anomic aphasia.

Recovery may be mediated by shifting of functions to the right hemisphere or to adjacent left hemisphere regions. Large, randomized trials have clearly indicated that patients who undergo formal speech therapy recover better than untreated patients do (Robey, 1998).Repeated practice in articulation and comprehension tasks traditionally has been used to stimulate improvement. Visual action therapy, which uses gestural expression; and treatment of aphasic perseveration, aims to reduce repetitive utterances.

MECHANISMS OF RECOVERY IN POST STROKE APHASIA

The greatest recovery in patients with aphasia due to stroke occurs during the first 3 months, but may be prolonged in global aphasics (1). Mechansisms of language recovery have been a subject of several studies PET and SPECT scans studies of language activation are advancements in understanding neuroanatomy of language recovery. Recovery in the first few days are due to the resolution of acute cellular derangements like oedema and restoration of blood flow to the ischaemic penumbra. The recovery of aphasias due to ischaemic stroke are better than the haemorrhagic stroke.

The finding of an excessive activation of homologous right sided brain regions in aphasics compared with normal subjects has suggested the role of non dominant hemispheric activation in the recovery process. Some studies have suggested that the preserved left hemispheric perilesional area plays an important role in the recovery of language function (20).

INFLUENCING FACTORS IN POST STROKE APHASIA

Factors determining the occurrence, severity and outcome of post stroke aphasia remains a subject of controversy. Increased incidence and prevelance of stroke in the elderly and prolonged duration of stroke survival contributes to the increased incidence of aphasia in the older population. Age related changes in the

language areas organization and influence on vascular pathology and infarct locations contributing to the poor outcome in older patients.

The next debated question is the sex difference in the functional organization of the brain for language. The functions of language are hypothesized to be highly lateralized in males and represented in both hemispheres in females and these have been supported by studies with functional MRI to map brain areas of language functions (21). The greater improvement of language in female aphasic patients has been accounted by the bilateral representation of language functions in female brain (22).

RESULTS

Total number of patients included in the study was 100. Out of this the maximum number of patients with stroke and aphasia were between 51 to 60 years age group in the males and 61 to 70 age group in the females. The mean age was 56.34years(Table 1).

Table 1:

AGE GROUP (in years)	NO. OF PATIENTS		TOTAL
	MALE	FEMALE	
< 40	7	2	9
41 – 50	15	4	19
51 – 60	27	4	31
61 – 70	19	9	28
>70	8	5	13
TOTAL	73	27	100

The majority were males – 73% and females contributing to 27%

Out of the 100 patients 64 were literates and 36 were illiterates. Among the males, 68.49% were literates and in females 51.85% were literates(Table 2).

Table 2:

LITERACY STATUS	MALE	PERCENTAGE	FEMALE	PERCENTAGE
LITERATE	50	68.49%	14	51.85%
ILLITERATE	23	31.50%	13	49.15%

Out of the 100 patients, 98 were right handed and 2 were left handed(Table 3).

Table 3:

HANDEDNESS	NO. OF PATIENTS
Right	98
Left	2

Among these, 40 patients had hypertension(33 – males , 7- females) while 23 were diabetics(17 – males , 6- females), 12 with Coronary Artery Disease(8 – males , 4-females), 4 patients had Rheumatic Heart Disease(1 – male , 3- females), 21 Smokers(males) and 19 Alcoholics(males)(Table 4).

Table 4:

RISK FACTORS	TOTAL NO. OF PATIENTS	MALES	FEMALES
Hypertension	40	33	7
Diabetes Mellitus	23	17	6
Coronary Artery Disease	12	8	4
Rheumatic Heart Disease	4	1	3
Smoker	21	21	0
Alcoholic	19	19	0

Out of the 100 patients, 82 patients had infarcts and 18 patients had haemorrhage.

Out of the 82 infarcts, 5 patients had haemorrhagic transformation(Table 5).

Table 5:

PATHOLOGY		NO. OF PATIENTS	PERCENTAGE
	Infarct	82	82%
	Haemorrhage	18	18%

The size of the infarcts in various types of Aphasia are given below

Table 6:

TYPE OF APHASIA	SIZE OF INFARCT(cm)							
	3.5X2	3x2.5	3x2	2x2.5	2x1	1x1.5	1x1	0.5x1
GLOBAL	18	13	20	10				
BROCA				1	6	1	5	2
WERNICKE	3	3	3	5	4			
TRANSCORTICAL MOTOR						1	2	
CONDUCTION						1		

The volume of bleed calculated in Aphasia patients with Haemorrhagic stroke are given below

Table 7:

TYPE OF APHASIA	VOLUME OF BLEED(ml)		
	15 – 20	10 - 15	< 10
GLOBAL	7	3	
BROCA		1	1
WERNICKE	3	3	

Most of the patients had severe neurological deficit as assessed by the NIHS score(Table 8).

Table 8:

NEUROLOGICAL DEFICIT	NIHS SCORE	NO. OF PATIENTS
Mild	1 – 7	8
Moderate	8 – 14	22
Severe	>15	70

Among the 100 patients majority had Global Aphasia - 61 patientss, Wernicke's Aphasia – 18 patients, Broca's Aphasia in 17 patients, Transcortical Aphasia in 3 patients, Conduction Aphasia in 1 patient(Table 9).

Table 9:

TYPE OF APHASIA	NO. OF PATIENTS	MALE	FEMALE
Global	61	45	16
Wernicke	18	15	3
Broca	17	10	7
Transcortical	3	-	3
Conduction	1	-	1
Total	100	73	27

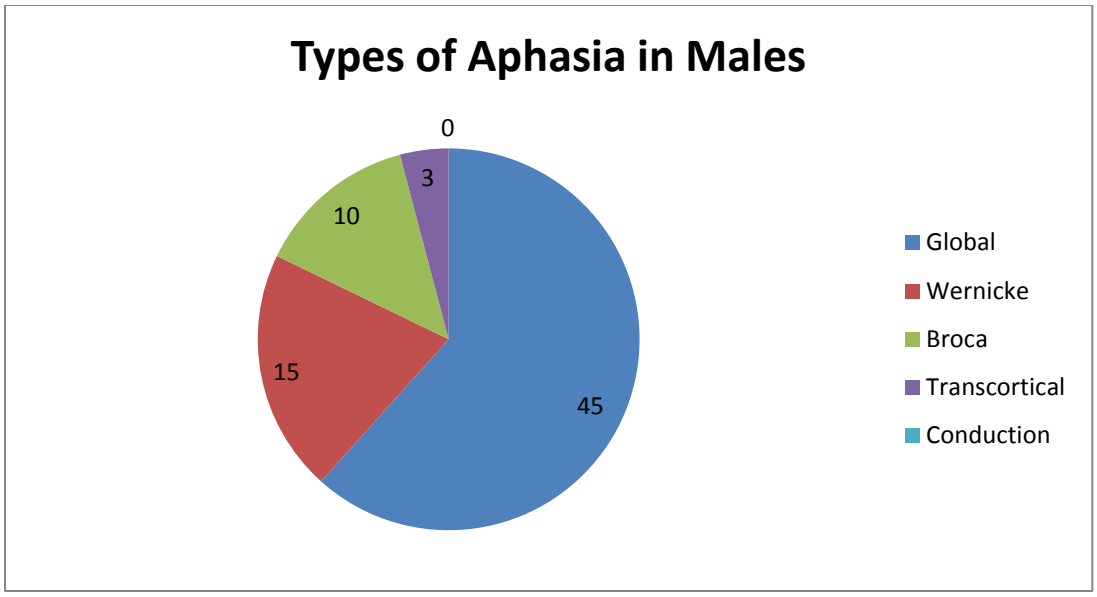


Fig 1:

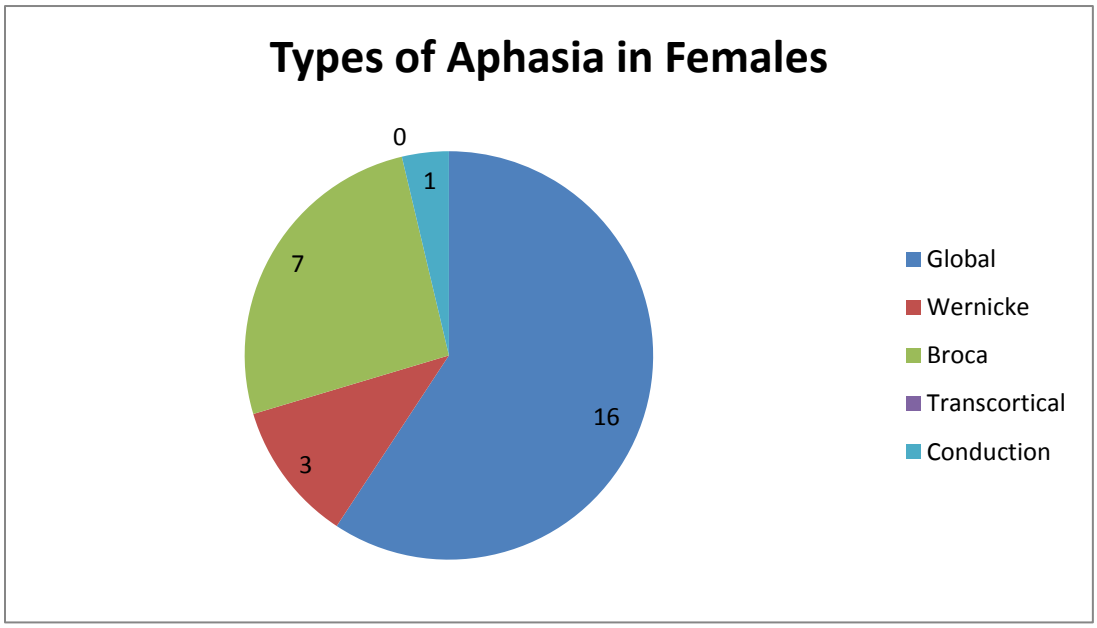


Fig 2:

Distribution in patients with different aphasia syndromes. The mean age of Global Aphasia patients was 57.13 and 59.22 in Wernicke's Aphasia patients. But the

mean age was younger in Broca's Aphasia patients 47.1 years. The mean age in Transcortical motor Aphasia was 50.3 years(Table 10).

Table 10:

Type of Aphasia	No. of patients	Mean Age(in years)
Global	61	57.13
Wernicke's	18	59.22
Broca's	17	47.1
Transcortical Motor	3	50.3

The relationship between age and initial aphasia type was analysed and found to be statistically insignificant $p>0.05$ (Table 11)

Table 11:

Relationship between age and initial aphasia type

Initial aphasia	**Age of patients (years)**	
	Mean	**SD**
Global	57.1	10.4
Wernicke	59.6	11.3

Broca	50.6	16.9
Transcortical	54.0	11.0
Conduction	61	0
'p'	0.2273 Not significant	

Among the risk factors in males, hypertension leads other risk factors, with Global Aphasia in 24 patients, Wernicke's Aphasia in 4 patients and Transcortical Aphasia in 2 patients. Diabetics with Global Aphasia were 10 patients, Wernicke's Aphasia in 4 patients, Broca's in 1 patient. Coronary Artery Disease with Global Aphasia were found in 6 patients, Wernicke's Aphasia in 2 patients and Transcortical Aphasia in 2 patients. Rheumatic Heart Disease with Broca's Aphasia in 1 patient. Smoking with Global Aphasia was found in 15 patients, Wernicke's in 3 patients and Broca's in 3 patients. Alcoholics with Global Aphasia was found in 14 patients, Wernicke's Aphasia in 3 patients and 2 patients with Broca's Aphasia.

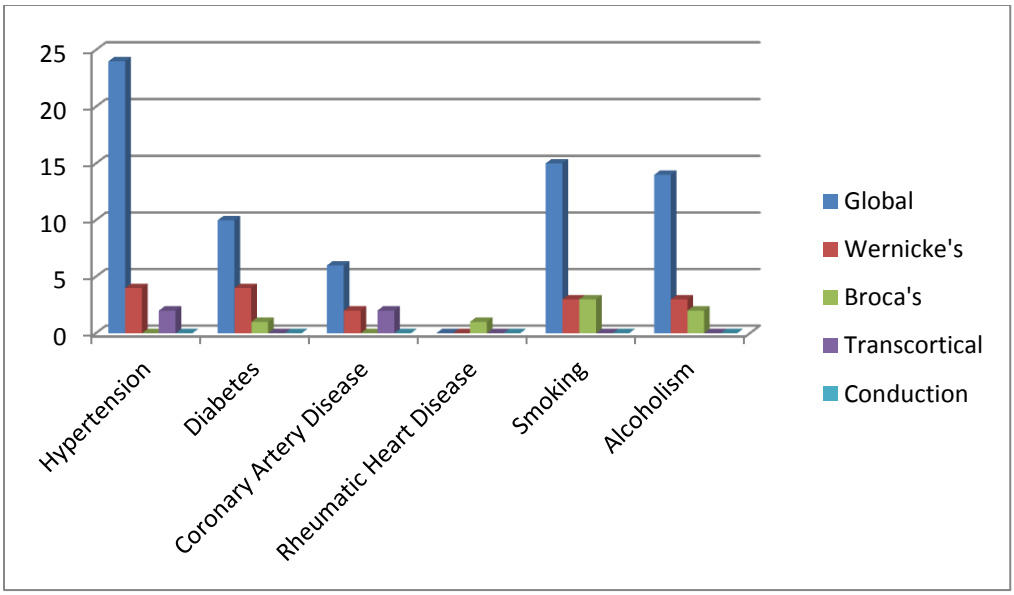


Figure 3 Risk Factors of Stroke and Types of Aphasia in male patients

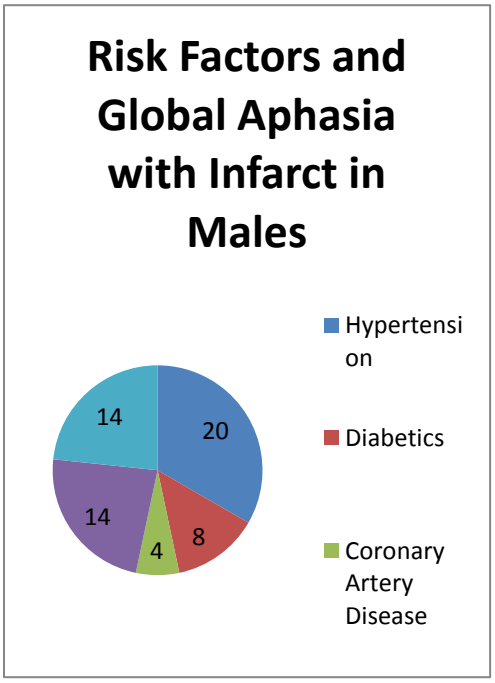


Fig 4

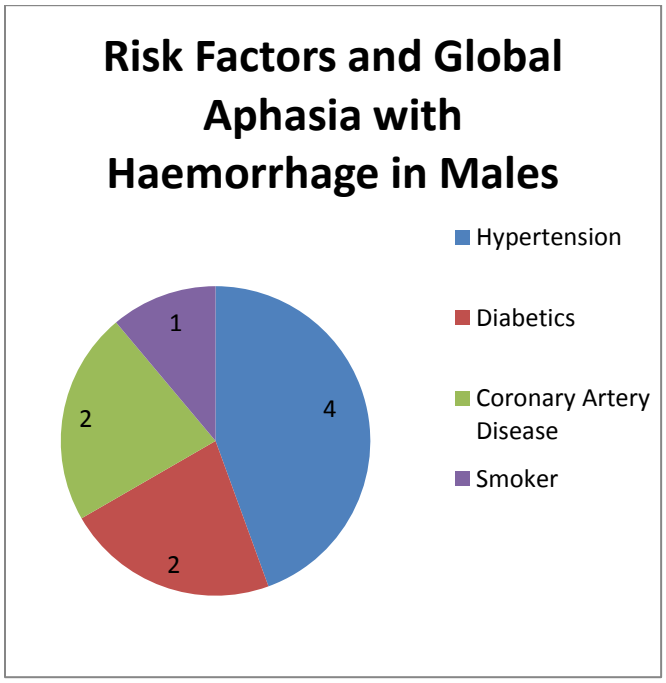


Fig 5:

Among the risk factors in females, Diabetics leads other risk factors, with Global Aphasia in 6 patients, Wernicke's Aphasia in 1 patients and Broca's Aphasia in 1 patients. Hypertension with Global Aphasia were 4 patients, Wernicke's Aphasia in 3 patients, Broca's in 2 patient and Conduction Aphasia in 1 patients. Coronary Artery Disease with Global Aphasia were found in 2 patients. Rheumatic Heart Disease with Global Aphasia in 1 patient and Broca's Aphasia in 2 patients.

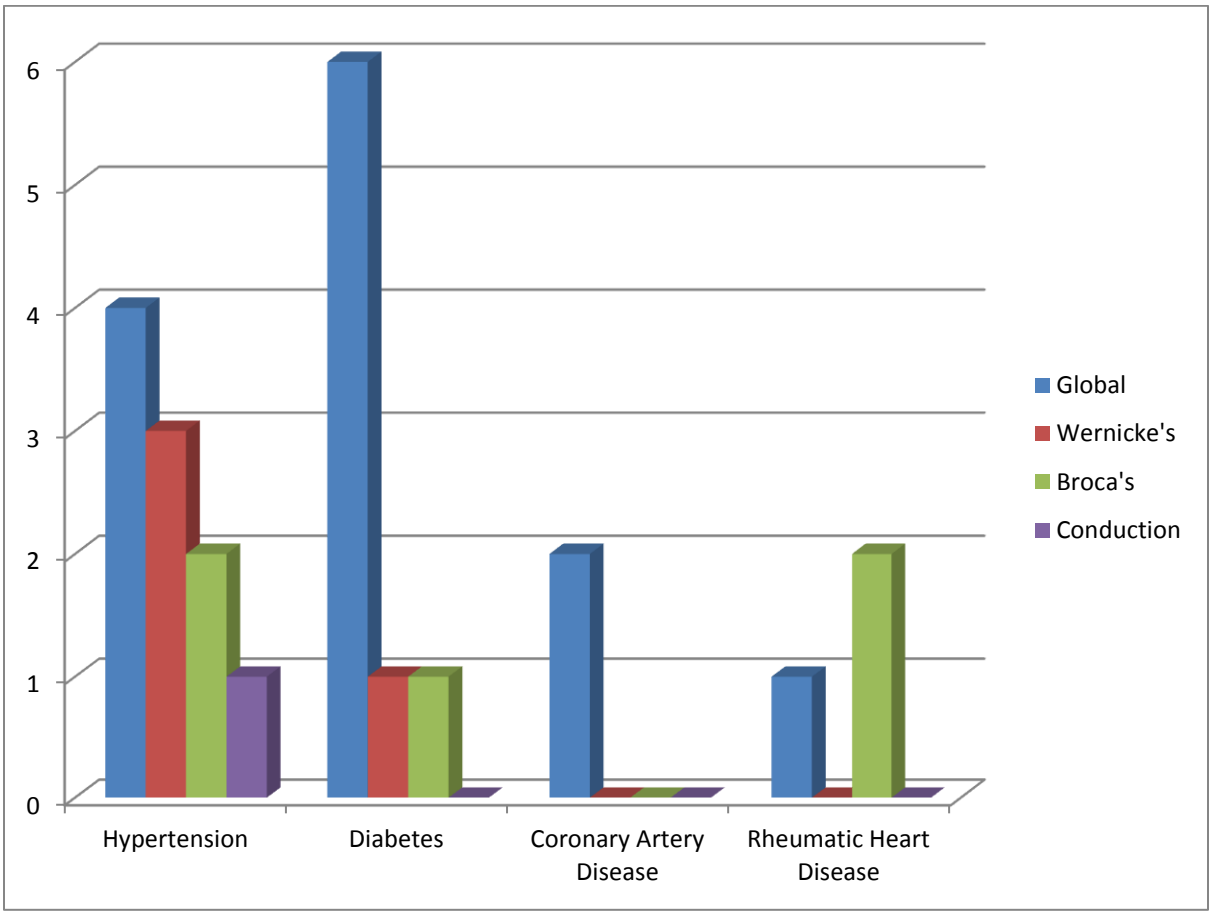


Fig 6: Risk Factors of Stroke and Types of Aphasia in female patients

Among the risk factors for Global Aphasia in females, diabetics were high – 6 patients followed by hypertensives – 4 patients and Coronary Artery Disease in 2 patients. All patients had Ischemic strokes.

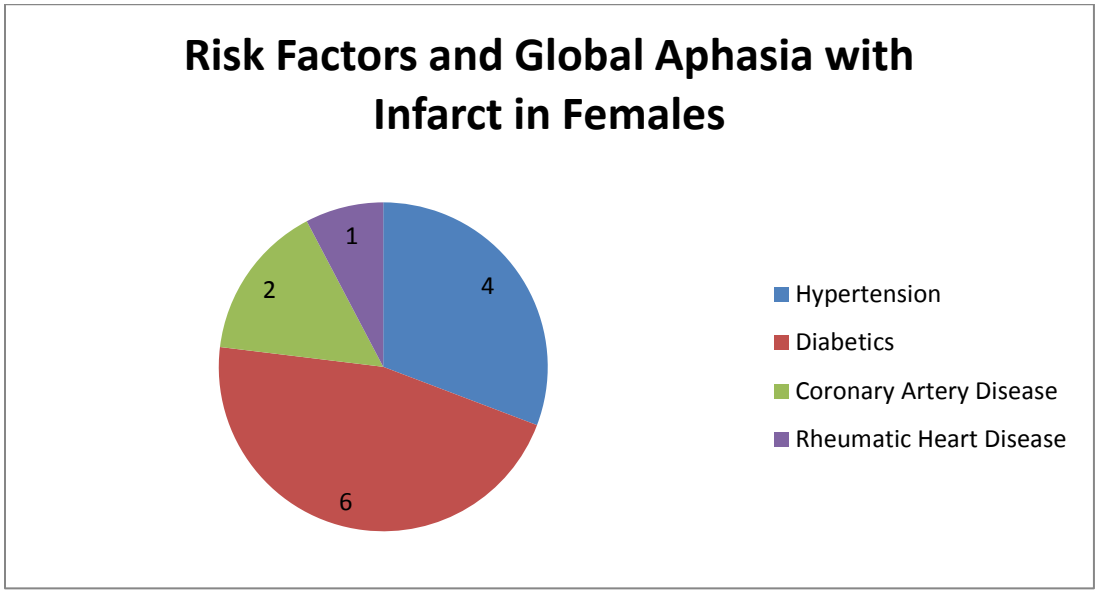
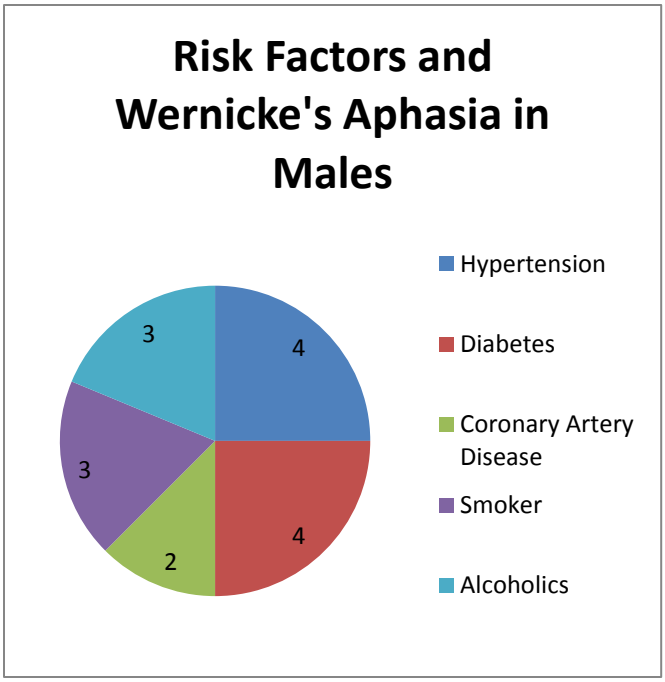


Fig 7

Among the risk factors for Wernicke's Aphasia in males, Diabetics and Hypertensives are equal – 4 patients each followed by smoker and alcoholics – 3 patients each, followed by Coronary Artery Disease in 2 patients. In females, Hypertension with Wernicke's Aphasia was found in 3 patients followed by Diabetes with Wernicke's Aphasia in 1 patient.

In males, hemorrhage with hypertension was one patient and other 3 infarcts .Infarcts and hemorrhage were equal in diabetics two each. In females all the 4 were infarcts.



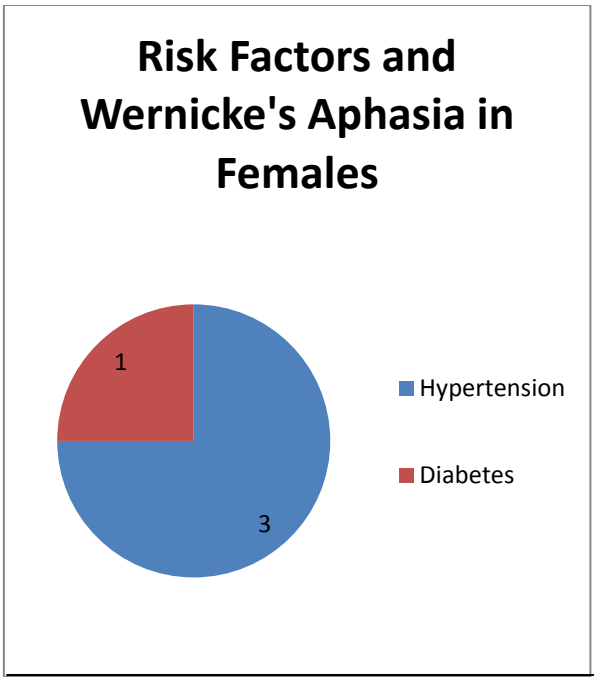


Fig 8

Fig 9

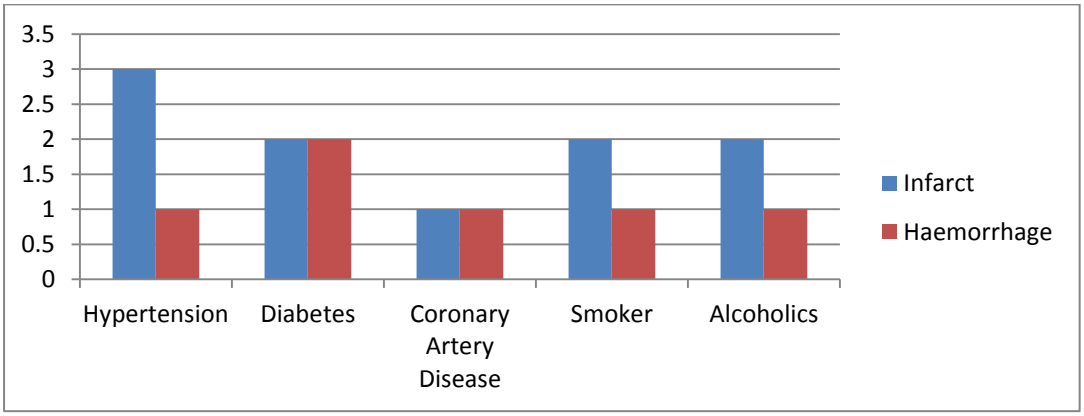


Figure 10: Risk Factors for Wernicke's Aphasia and tye of Lesion in Males

Among the female patients with Wernicke's Aphasia all the 4 patients had infarcts, 3 with Hypertension and 1 with Diabetes.0

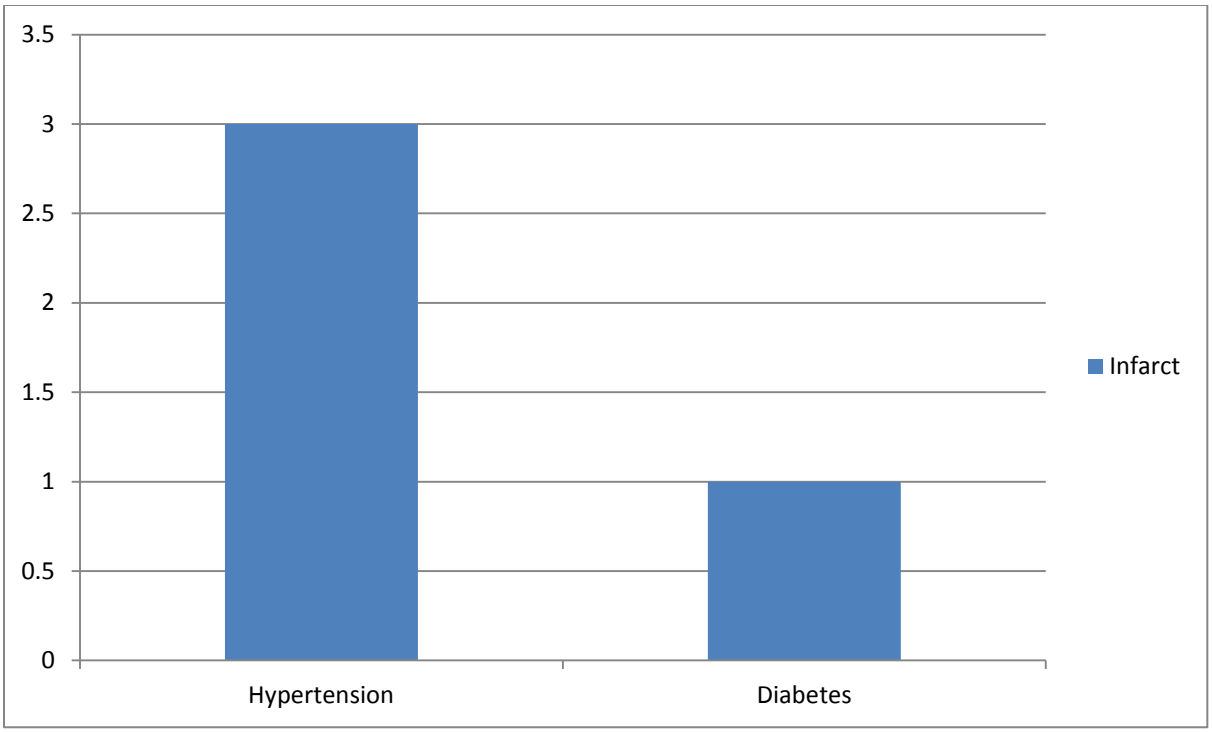


Figure 11: Risk Factors for Wernicke's Aphasia in Females

Among the risk factors for Broca's Aphasia in males, Diabetes in 1 patient. Smoker – 3 patients and alcoholics – 2 patients.In females, Hypertension with Broca's Aphasia was found in 2 patients followed by Diabetes in 1 patient, Rheumatic Heart Disease with Broca's Aphasia in 2 patients.

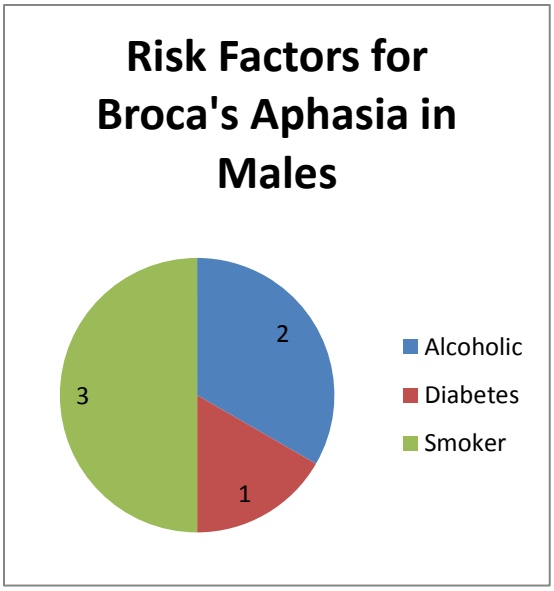


Fig 12

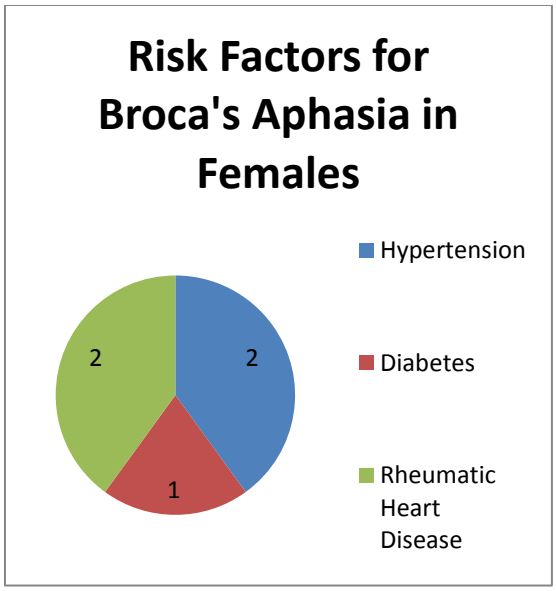


Fig 13:

Among the male patients with Broca's Aphasia, all the 6 patients had Infarcts with 1 patient being Diabetic, 3 Smokers and 2 Alcoholics. Among the female patients with Broca's Aphasia, 2 were hypertensives, 1 had infarct and 1 had haemorrhage and other patients with diabetes and rheumatic heart disease had infarcts.

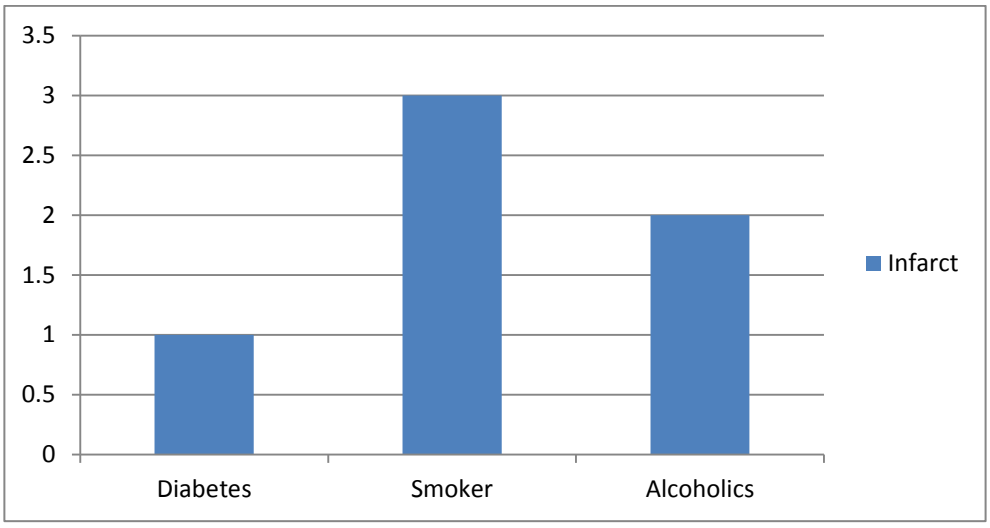


Figure 14: Risk Factors for Broca's Aphasia with Infarct in Males

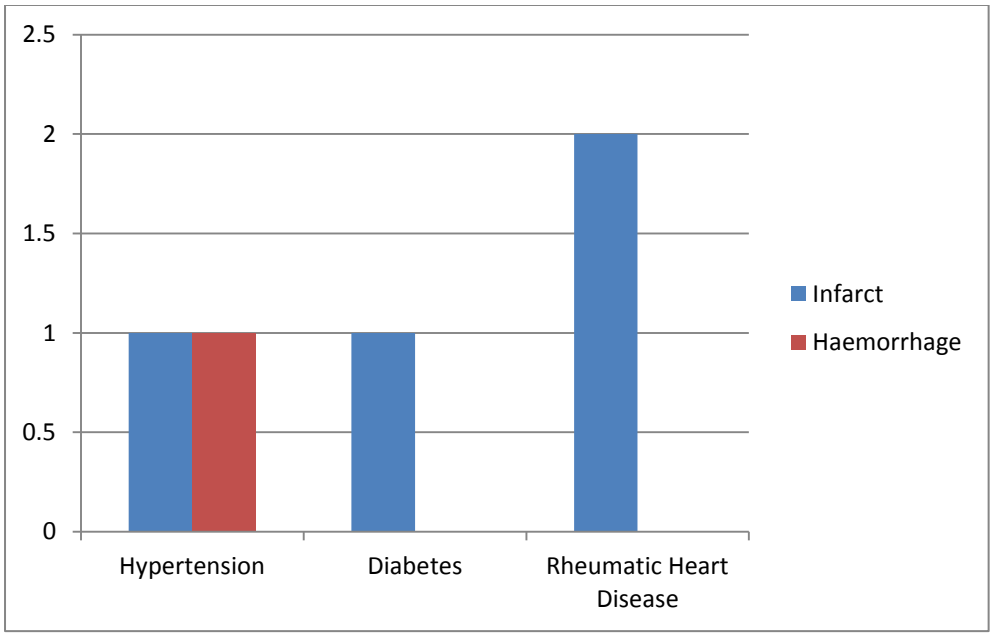


Figure 15: Risk Factors for Broca's Aphasia and type of Lesion in Females

Transcortical Motor Aphasia was found in 3 male patients, out of them 2 had Hypertension and Coronary Artery Disease and they all had Infarcts. Conduction Aphasia was found in 1 female patient with Hypertension and Infarct.

FOLLOW UP

Among the 100 patients enrolled, 40 were lost to follow up because of premature deaths/ absence of follow up.

Among the 60 patients followed up, 51 were male patients and 9 were female patients. Out of the 9 female patients, 8 had Infarcts with 4 Global Aphasia, 2 Broca's and 2 Wernicke's Aphasia and 1 had Haemorrhage with Broca's Aphasia. Among the 51 male patients, 36 patients had Global Aphasia with 27 Infarcts and 9

Haemorrhage, Wernicke's Aphasia in 8 patients, all being Infarcts and Broca's Aphasia in 7 patients with all 7 Infarcts.

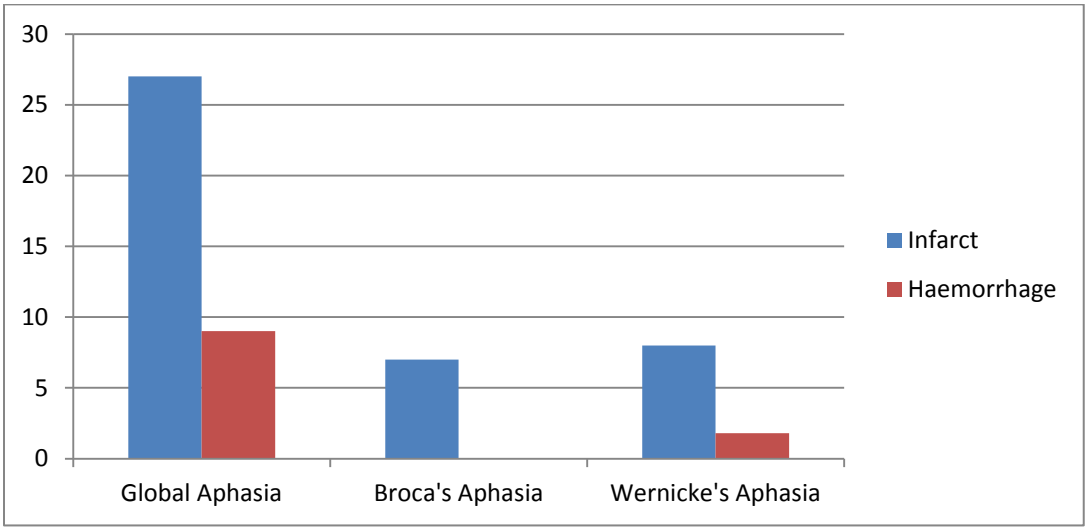


Figure 16: Aphasia and Type of Lesions in followed up males

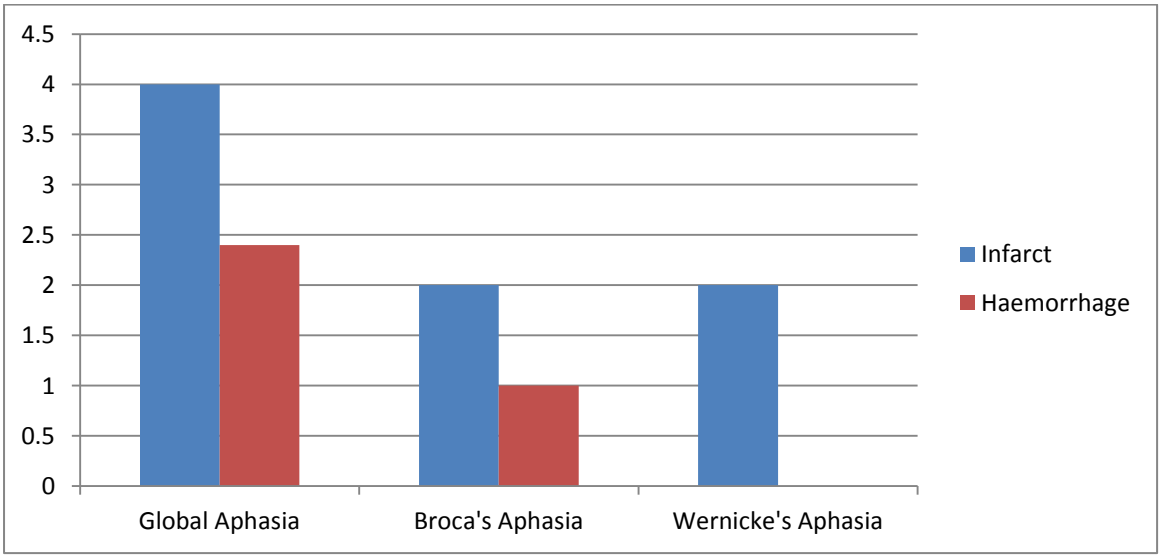


Figure 17: Aphasia and Type of Lesions in followed up females

EVOLUTION OF APHASIA

Out of the 9 female patients followed up, 2 Wernicke's remained as Wernicke's, 1 Broca's Aphasia evolved to Transcortical Motor, 2 Broca's remained as Broca's, 3 Global Aphasia evolved to Broca's, 1 Global remained as Global.

Out of the 51 male patients, Global Aphasia constituted 36 patients out of which 12 Global Aphasia evolved to Broca's Aphasia with Infarct in 9 patients and Haemorrhage in 3 patients, 24 remained as Global Aphasia with 6 Haemorrhage and 18 Infarcts. 8 Wernicke's Aphasia remained as Wernicke's Aphasia. Out of 7 Broca's Aphasia patients 3 evolved to Transcortical Motors and rest remained as Broca's(Table 12).

Table 12:

APHASIA EVOLUTION	MALES		FEMALES		MEAN AGE
	NO. OF PATIENTS	PERCENTAGE	NO. OF PATIENTS	PERCENTAGE	
WITHOUT EVOLUTION	36	70.8%	5	55.5%	57.12
EVOLUTION TO OTHER TYPE	15	29.41%	4	44.44%	46.57

The relation between age and evolution of aphasia was found to be statistically significant p<0.001(Table 13)

Table 13: **Age and evolution of Aphasia**

Type of case	**Age of patients (years)**	
	Mean	**SD**
Cases with evolution	61.1	10.2
Cases without evolution	53.2	12.2
'p'	**0.0011** **Significant**	

Out of the 51 male patients, 15(29.41%) patients evolved to other type of Aphasia. In the females, out of the 9 patients, 4(44.44%)patients evolved to other type, with the percentage being higher in females. The mean age of patients with evolution was 46.57 and in the patients without evolution the mean age was 57.12 years. The mean age of male patients with evolution was 46.3 and in females the mean age was 47.5 years.

The relation between age and evolution of aphasia was statistically significant (p<0.001)(Table 14)

Table 14:

Age and evolution

Type of case	**Age of patients (years)**	
	Mean	**SD**
Cases with evolution to other type	46.2	11.7
Cases without evolution	56.5	11.0
‘p’	**0.0016** **Significant**	

The relation between sex and evolution was not significant (p >0.005)

Table 15:.

Sex and evolution

Type of case	Sex of patients			
	Male		Female	
	No.	%	No.	%
Cases with evolution to other type(19)	15	78.9	4	21.1
Cases without evolution (41)	37	90.2	4	9.8
'p'	0.2114 Not Significant			

GLOBAL APHASIA AND TEST SCORES

Patients with Global Aphasia who evolved to Broca's Aphasia had good initial aphasia Quotient scores than the patients not evolved. The mean AQ score in evolved type was 47.1 and 11.46 in the unevolved patients(Table 16).

Table 16:

CASE NO.	T1(1^{ST} WEEK)	T2(12 WEEKS)	T3(24 WEEKS)	INITIAL TYPE OF APHASIA	FINAL EVOLUTION
3	2.8	2.8	11.6	Global	Global
4	2.8	11.2	24	Global	Global
7	14.4	39.6	66	Global	Broca's
8	24	42.2	62.4	Global	Broca's
9	2.8	2.8	4.2	Global	Global
12	2.8	2.8	4.2	Global	Global
14	7.2	30.4	51.6	Global	Broca's
15	2.8	3.6	3.6	Global	Global
17	12	35.6	56.4	Global	Broca's
19	12	37.4	57.6	Global	Broca's
27	7.2	36	55.6	Global	Broca's
29	2.8	4.2	16.2	Global	Global
32	13.2	35	57.6	Global	Broca's
33	2.8	3.6	4.2	Global	Global
41	2.0	10.2	22.2	Global	Global

43	8.2	36	52	Global	Broca’s
44	2.8	5.2	17.2	Global	Global
45	13.2	48.8	60	Global	Broca’s
46	7.4	30.8	52	Global	Broca’s
47	3.6	4.2	4.2	Global	Global
48	2.0	12.2	22.2	Global	Global
53	2.8	8.2	24.2	Global	Global
55	13.2	39	62	Global	Broca’s
60	2.8	12	16.2	Global	Global
61	12	42	65.6	Global	Broca’s
63	2.8	11.6	15	Global	Global
67	13.4	28.4	54.4	Global	Broca’s
68	2.8	7.2	9.2	Global	Global
72	2.8	9.2	20.2	Global	Global
75	12	41.6	64	Global	Broca’s
76	7.4	33.6	54	Global	Broca’s
77	2.8	9.6	20.2	Global	Global
79	2.8	7.2	20.2	Global	Global
80	2.8	3.6	5.6	Global	Global
85	2.8	8.2	22	Global	Global

93	2.8	2.8	6.8	Global	Global
94	3.6	2.8	25	Global	Global
98	2.8	9.6	20.2	Global	Global
99	2.8	7.6	11.6	Global	Global
100	2.8	2.8	18.6	Global	Global

BROCA'S APHASIA AND TEST SCORES

4 patients evolved to Transcortical Motor Aphasia and 6 patients remained as Broca's Aphasia. The mean AQ score in evolved patients was 36.05 and in the unevolved 23.06(Table 18).

Table 18:

CASE NO.	T1(1^{ST} WEEK)	T2(12 WEEKS)	T3(24 WEEKS)	INITIAL TYPE OF APHASIA	FINAL EVOLUTION
13	27.94	50.8	75.2	Broca's	Transcortical Motor
22	60.4	65.2	89.6	Broca's	Transcortical Motor

28	25	34	43.6	Broca's	Broca's
39	58	65.2	89.6	Broca's	Transcortical Motor
49	29.04	54.8	65.2	Broca's	Transcortical Motor
51	22	36.4	50.2	Broca's	Broca's
69	23	32.4	44.8	Broca's	Broca's
73	21.6	25	34	Broca's	Broca's
83	22	36.4	51.2	Broca's	Broca's
90	21	33	49.2	Broca's	Broca's

WERNICKE'S APHASIA AND TEST SCORES

All the patients remained as Wernicke's, but the final scores were better than the initial scores. The mean AQ score was 13.82(Table 19).

Table 19:

CASE NO.	T1(1^{ST} WEEK)	T2(12 WEEKS)	T3(24 WEEKS)	INITIAL TYPE OF APHASIA	FINAL EVOLUTION
16	22.8	33	42.4	Wernicke's	Wernicke's
35	18.4	21.6	30	Wernicke's	Wernicke's
36	20.4	24	28.4	Wernicke's	Wernicke's
57	22	28	30	Wernicke's	Wernicke's
59	21.8	30	37.2	Wernicke's	Wernicke's
70	25	30.8	32.4	Wernicke's	Wernicke's
84	21.6	28.4	38.8	Wernicke's	Wernicke's
87	20.8	26	34.8	Wernicke's	Wernicke's
95	20.2	29	42.8	Wernicke's	Wernicke's
97	18.4	30.4	32.8	Wernicke's	Wernicke's

The Aphasia Quotient was better in patients whose aphasia evolved to other type than the patients whose aphasia did not show evolution(Table 20).

Table 20:

TYPE OF APHASIA	NO. OF PATIENTS	MEAN AQ EVOLVED	MEAN AQ NOT EVOLVED	MEAN AQ (FINAL SCORE – INITIAL SCORE)
Global	40	47.1	11.46	24.83
Broca's	10	36.05	23.06	28.26
Wernicke's	10	-	13.82	13.82

The relation between age group and aphasia quotient was not significant ($p>0.5$) initially ,but at the end of six months it was highly significant ($p<.001$)(Table 21

Table 21: Age group and Aphasia quotients

Age group	**Aphasia quotient**			
	Initial		**At the end of 6 months**	
	Mean	**SD**	**Mean**	**SD**
Upto 40 years	27.9	19.2	66.0	18.0
41-50 years	11.4	12.3	33.1	19.4

51-60 years	15.8	14.5	31.1	20.0
61-70 years	12.7	18.2	32.2	22.0
>70 years	15.9	15.2	26.8	18.4
'p'	0.1099 Not significant		**0.0009** **Significant**	

The relation between sex and aphasia quotient at the end of six months was statistically significant ($p<0.001$)(Table 22)

Table 22: **Sex and aphasia quotients**

Sex	**Aphasia quotient**			
	Initial		**At the end of 6 months**	
	Mean	**SD**	**Mean**	**SD**
Male	14.3	14.3	33.4	22.1
Female	17.8	20.1	52.5	19.4
'p'	0.341 Not significant		**0.0249** **Significant**	

The relation between NIHSS score and aphasia quotient was significant initially and at the end of six months ($p<0.001$)(Table 23)

Table 23: **NIHS score and aphasia quotients**

NIHS score	**Aphasia quotient**			
	Initial		**At the end of 6 months**	
	Mean	**SD**	**Mean**	**SD**
Mild	33.4	17.8	59.9	25.9
Moderate	15.9	7.7	49.0	10.1
Severe	12.7	16.1	26.1	20.5
'p'	**0.0008 Significant**		**<0.0001 Significant**	

The relation between aphasia type and aphasia quotient was statistically significant($p<0.001$)(Table 24)

Table 24: **Aphasia type and aphasia quotients**

Aphasia type	**Aphasia quotient**			
	Initial		**At the end of 6 months**	
	Mean	**SD**	**Mean**	**SD**
Global	5.5	4.4	14.0	7.6
Wermicke	21.0	1.9	35.0	5.2
Broca	32.6	15.7	54.2	7.9
Transcortical	51.1	0.6	79.9	11.9
Conduction	81.7	0	-	-
'p'	**<0.0001 Significant**		**<0.0001 Significant**	

An analysis of correlation efficient between age and aphasia quotient ,it was found to be negatively correlated. Correlation efficient between the area of infarct and

initial aphasia quotient was negatively correlated. The correlation efficient between hemorrhage volume and final aphasia quotient was found to be negatively correlated(Table 25).

Table 25: **CORRELATION**

Correlation coefficient between	**Initial Alphasia quotient**	**Final Alphasia quotient**
Age	-0.1699	-0.4575
NIHSS score	-0.3709	**-0.6641 Correlated**
Infarct area	**-0.6651 Correlated**	-0.4709
Haemorrhage volume	-0.4634	**-0.5457 Correlated**

CORRELATION OF IMAGING WITH APHASIA

All Global Aphasia patients had large infarct involving left frontal, parietal and temporal lobes, both cortical and sub cortical regions including the Broca's, Wernicke's and the adjacent areas. Out of the 40 Global Aphasia patients, 15 evolved into Broca's and 25 remained as Global. The size of the infarcts in patients who evolved into Broca's are given as follows

Table 26:

SIZE OF THE INFARCT(cm)	NO. OF PATIENTS
3 x 2.5	6
2 x 2.5	3
3 x 2	3

The size of the infarcts in patients who remained as Global are given as follows

Table 27:

SIZE OF THE INFARCT(cm)	NO. OF PATIENTS
3.5 x 2	8
3 x 2	10
2 x 2.5	4

Patients with Broca's Aphasias showed infarct in the left frontal lobe involving the Broca's cortical area and adjacent sub cortical areas. In patients who evolved into Transcortical Motor, the size of the infarcts are given as follows

Table 28:

SIZE OF THE INFARCT(cm)	NO. OF PATIENTS
0.5 x 1	2
1 x 1.5	

In patients who remained as Broca's , the size of the infarcts are given as follows

Table 29:

SIZE OF THE INFARCT(cm)	NO. OF PATIENTS
1 x 1	3
2 x 0.5	1

Patients with Wernicke's Aphasia had infarct in left temparo parietal region including Wernicke's, supra marginal gyrus, cortical and sub cortical regions. The size of the infarcts in patients with Wernicke's Aphasia are given as follows

Table 30:

SIZE OF THE INFARCT(cm)	NO. OF PATIENTS
3.5 x 2	3
3 x 2	2
2 x 2.5	4
2 x 1	1

The patients who had small size infarcts had better recovery than the patients with large size infarcts. The mean size of infarcts was 5.12 cm^2

The volume of bleed in Hemorrhagic stroke calculated by the formula abc/2 in the different types of aphasia are given below

Table 31:

VOLUME OF BLEED	NO. OF PATIENTS	APHASIA TYPES	
		INITIAL	FINAL
15 – 20 ml	3	Global	Global
10 – 15ml	2	Broca's	Broca's
	1	Global	Broca's
< 10ml	2	Global	Broca's

Patients who had bleed less than 10ml had better recovery than the patients with large volume of bleed.

The mean volume of hemorrhage was 14.9 ml^3

DISCUSSION

The mean age group of patients in this study was 56.34 years with the maximum number of patients in the 5^{th} to 6^{th} decade. Majority of the patients(73%) were males and right handed. In Indian studies done by Paithankar et al (22) the mean age was 71.3 and maximum number of male patients. Study conducted by O.Godefroy et al (23) France on characteristics of aphasia in acute stroke reported the mean age of patients 62 with male predominance and right handed. This data is consistent with other studies which have reported higher prevelance of stroke in elderly age group and in males.

Global Aphasia constituted as 61% and Wernicke's, Broca's and Transcortical Aphasias into 40%. The data is consistent with the study done by O.Godefroy et al who reported Global Aphasia in 50% of the patients, whereas studies done by Pederson et al have reported lower incidence. Broca's Aphasia constituted 17% of the cases in this study and Wernicke's Aphasia 18% in this study. This is consistent with the other studies done by McDermott et al (24) and Copenhagen aphasia study (25) which reported Broca's 15% and 12%, Wernicke's 18% and 16% respectively. In this study 3 patients had Transcortical Aphasia and 1 had Conduction Aphasia. This data is lower than in the studies quoted above which may be explained by referral bias.

There was no significant age difference among the patients with different types of aphasia in this study, though the patients with Broca's aphasia were slightly younger (mean age – 47.1years) than patients with Wernicke's(59.2years) and Global Aphasia(57.13years). This is consistent with Kertesz et al's (26) study which reported Broca's aphasia in younger age group than others and related the slight age difference to pathophysiological factors favouring embolic strokes in the anterior division of middle cerebral artery territory. Godefroy et al reported that age did not significantly differ across various aphasias except Conduction aphasias in younger patients and Subcortical aphasias in elder patients. Studies done by Pashek and Holland (27) reported better recovery in younger patients. Studies done by Sarno (28) and Levita using a subjective and functional assessment of language failed to show significant changes in correlation with age, education and initial performance.

There was no significant sex difference among various aphasias in this study. This is consistent with studies by Kertesz ae al and Godefroy et al. he spontaneous recovery of language in patients with aphasia studied by Lendren W et al revealed that age, sex and type of aphasia were not related to the amount of improvement (29).

Majority of the patients had some improvement in their aphasia quotient. In this study 19 out of the 60 patients(30.1%) had a change in aphasia type. Among

the 40 patients with global aphasia 15 evolved to Broca's aphasia and the rest remained as global. Among the 10 Broca's aphasia patients, 4 evolved into transcortical motor and the rest remained as Broca's. all the wernicke's aphasias remained as Wernicke's. This is consistent with the data in the Copenhagen aphasia study which concluded that non fluent aphasia could evolve into a fluent aphasia whereas a fluent aphasia never evolve into a non fluent one. Andrew Kertesz arid, McCabe noted good recovery in Broca's aphasia and Vignolo (30) noted poor outcome in his study. In our study Broca's aphasia had a good outcome.

Kertesz and McCabe noted a bimodal pattern of recovery in Wernicke's aphasia ie patients with initially low scores had poor outcome and those with high scores had good outcome. All the Wernicke's patients in our study had low initial scores and showed fair recovery.

The relation between age and evolution of aphasia was statistically significant. More females (44.44%) had evolution of aphasia compared to males(29.41%) suggesting that language outcome is better in females.

Pizzamiglio (31) et al studied the sex differences in aphasia recovery and reported females with global aphasia had greater improvement than males, although no initial significant sex difference was found. The better recovery in females was explained by the bilateral representation of the language in the female

brain compared to the male brain. Gender differences in aphasia studied by Heir et al showed no gender differences in aphasia due to hemorrhagic stroke, but infarct more frequent in women than men (32).

In our study global aphasia was higher in male patients(65%) than female patients(48%). Broca's aphasia was higher in female patients(25.92%) than male patients(13.69%). Wernicke's aphasia was also higher in male patients(20.5%) than female patients(11.11%).

The change in aphasia quotient(final AQ – initial AQ) was significantly higher in patients whose aphasia evolved than the patients whose aphasia not evolved in this study. McDermott et al reported a significant changes in aphasia scores in patients whose aphasia type evolved into another and the study also concluded that initial severity was one of the factors predicting aphasia recovery. Sarno, Silverman and Sands (33) noted good outcome in patients who had high initial scores. Similar results was noted by Kertesz and McCabe et al. The relation between aphasia quotient and NIHSS, aphasia quotient and aphasia type, sex and aphasia quotient were statistically significant in our study.

Studies done by Vijayaraghavan and Natarajan et al on 16 stroke patients with aphasia noted considerable recovery in auditory verbal comprehension,

especially in auditory word recognition for numbers, bodyparts and objects, in patients with global and Wernicke's and low aphasia scores (34).

Among the risk factors, hypertension leads followed by diabetes mellitus, coronary artery disease and rheumatic heart disease. In males smoking and alcoholism constitute major risk factor.

In this study, increase in aphasia quotient in the first 3 months following the stroke was higher than the subsequent months, indicating that maximum recovery of language functions occurred in the first 3 months. This is consistent with the studies by Kertesz et al and McDermott et al in which they attributed the improvement to the resolution of acute ischemic changes like perilesional edema and restoration of blood flow to the ischemic penumbra.

Correlating the change in AQ to the initial severity of stroke as measured by the NIHS scale, there was asignificant negative correlation.

In this study, ischemic stroke constituted 82% and haemorrhagic stroke 18%. The data is consistent with the Indian studies done by Kulshrestha M, Vidyanand(35) on analysis of risk factors of stroke in North India in 157 patients reported ischemic stroke more frequent than haemorrhagic stroke(71 vs 29). The study also revealed hypertension, the most prevalent risk factor in stroke followed by diabetes mellitus cum hypertension. This data is also consistent with our study

where hypertension is the major risk factor followed by diabetes mellitus, coronary artery disease and smoking and alcoholism in males.

The size of the infarct had significant correlation with outcome of aphasia. Patients with small sized infarcts had a better outcome. The volume of bleed also had similar significant correlation with outcome of aphasia.The correlation efficient between infarct and initial aphasia quotient and correlation efficient between hemorrhage volume and final aphasia quotient were found to be negatively correlative.

There was good correlation with the anatomical location of the lesion and CT scan. Separate lesion sites for Broca, Wernicke's, Conduction and transcortical motor aphasia were demonstrated on CT scan. The lesion sites were consistent with Geschwind's concept of aphasia (36).

Wernicke's aphasia was associated with posterior lesions and Broca's and Global with anterior lesions. The data was consistent with the studies done by Benson et al and Kertesz et al (37) on radio nuclide brain scans. The study by Kertesz et al showed central lesions in conduction aphasia along with anterior and posterior lesions and extensive lesions in global aphasia. Similar results were shown by the angiograms, radio nuclide brain scans and CT scans in the study done by Yarnell, Monroe and Sobel (38).

Studies by Margaret Naeser and Robert Hayward (39) with CT brain showed that the lesions in Broca's aphasia was large involving the Broca's cortical and subcortical areas. In Wernicke's aphasia the lesion extended into the subcortical areas along with the Wernicke's cortical area and also the supra marginal angular agyrus. Patients with conduction aphasia had lesions deeper to the Wernicke's area compatible with the involvement of arcuate fasciculus. The above findings are compatible with the Kertesz radionuclide brain scan study. Dominant frontal lobe lesions associated with Transcortical motor aphasia and did not involve Broca's area. Large lesions involving the perisylvian areas were associated with Global aphasia. Large lesions involving cortical and subcortical areas of frontal, parietal and temporal lobes were noticed by Kertesz and Yarnell and also noted poor recovery in these patients.

Study by Mohr et al (40) in 30 patients with detailed autopsy, EMI scan and arteriogram data found that infarct in Broca's area and its surrounding deep areas causes mutism followed by rapidly improving dyspraxia and effortful articulation without significant disturbance in language function. The infarct in Broca's area does not cause Broca's aphasia. The Broca's aphasias is associated with the larger infarct involving operculum, Broca's area, insula and adjacent areas in the territory of upper division of left middle cerebral artery.

CONCLUSION

- Global aphasia is the most common aphasic syndrome in acute stroke patients followed by Wernicke's aphasia, Broca's aphasia, Transcortical motor aphasia and Conduction aphasia. Age and sex did not differ significantly in various types of aphasia.
- Recovery in patients with aphasia due to stroke is a dynamic process with evolution of aphasia from one type to another. There was some improvement in language function as reflected by the aphasia quotient in most of the patients followed up. The type of aphasia change to a less severe form in 30.1%.
- Patients with Global aphasia evolved into Broca's aphasia in 15 patients out of 40 patients. Out of the 10 Broca's aphasia patients, 4 evolved into Transcortical motor aphasia. Significant improvement was noted after 12^{th} week of stroke.
- Hypertension and Diabetes were the major risk factors.
- There was a good correlation with the clinical – anatomical location of lesion and imaging.

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PROFORMA

1) Name of the patient :

2) Age :

3) Sex :

4) Literacy Status : Literate/Illiterate

5) Handedness :

6) InPatient Number :

7) Occupation :

8) Date of Admission :

9) Duration of Illness :

10) Past similar episodes :

11) Comorbid Illness : DM/HT/CAD/RHD/ SMOKER/ALCOHOLIC

12) Details of treatment :

13) Family history : Yes/No

14) Biochemical profile :

15) Radiological findings : CT/MRI

16) Follow up after 12th week :

24th week :

- Static
- Improving

17) Associated Deficits :

18) NIHS Score :

19) LOBAR Functions :

- Frontal :
- Temporal :
- Parietal :
- Occipital :

20) WAB Score :

- During Admission :
- 12 weeks :
- 24 weeks :

21) Type of Aphasia :

22) Evolution :

WESTERN APHASIA BATTERY (TAMIL VERSION)

3. Sequential Commands:

1. கையை தூக்கு (2)
2. கண்ணை மூடு (2)
3. நாற்காலியைக் காண்பி (2)
4. ஜன்னலைக் காண்பித்து விட்டு கதவைக் காண்பி. (4)
5. பேனாவையும் புத்தகத்தையும் காட்டு (4)
6. பேனாவால் புத்தகத்தைக் காட்டு. (8)
7. புத்தகத்தால் பேனாவைக் காட்டு (8)
8. பேனாவால் அளவுகோலைக் காட்டு (8)
9. புத்தகத்தால் அளவுகோலைக் காட்டு (8)
10. புத்தகத்தின் மேல் பேனாவை வைத்து என்னிடம் கொடு (14)
11. பேனாவின் பக்கத்தில் அளவுகோலை வைத்து புத்தகத்தை அதன் மேல் வை. (20)

Max. Score: 80
Patient's Score:

III REPETITION

	Max. Score	Patient's Score
1. பஸ்	2	
2 மூக்கு	2	
3 கண்	2	
4 ஜன்னல்	2	
5 வாழைப்பழம்	4	
6 கைக்கடிகாரம்	4	
7. நாற்பத்தி ஐந்து	4	
8. தொண்ணூற்று ஐந்து சதவிகிதம்	6	
9 இரண்டு மணி ஐம்பத்து ஐந்து நிமிடங்கள்	10	
10 மணி அடித்துக் கொண்டு இருக்கிறது	8	
11 கடலோரத்தில் பந்து உருளுது புரளுது	10	
12. மின்னுவதெல்லாம் பொன்னல்ல	6	
13 யானைக்கொரு காலம் வந்தால் பூனைக்கொரு காலம் வரும்	10	
14 நான் கடைக்குப் போய் மிட்டாய் வாங்கினேன்	10	
15 சென்னை பாரிஸ் கார்னரில் இருந்து முப்பது கிலோமீட்டர் தூரத்தில் மகாபலிபுரம் உள்ளது	20	
	100	

Max. Score: 100
Patient's Score:

IV. NAMING

a) Object Naming:

1. கைக்கடிகாரம் 2. பேனா 3. புத்தகம் 4. காசு 5. சாவி.
6. அளவுகோல் 7. டார்ச் லைட் 8. இன்ச் டேப் 9. மணி பர்ஸ் 10. சோப்பு டப்பா
11. தீப்பெட்டி 12. டூத் பிரஷ் 13. பூட்டு 14. சீப்பு 15. ஸ்பூன்
16. இங்க் பாட்டில் 17. கேசட் 18. விக்ஸ் டப்பா 19. பவுடர் டின் 20. பல்பு

Max. Score: 60
Patient's Score:

b) Word Fluency: உனக்குத் தெரிந்த மிருகங்களீ பெயரைக் கூறு.

Max. Score: 20
Patient's Score:

c) Sentence Completion:

1. பாலின் நிறம் .....
2. குளத்தில் .....
3. பேனாவினால் .....
4. பொங்கல் கொண்டாடும் மாதம் .....
5. காற்றுள்ள போதே .....

Max. Score: 10
Patient's Score:

d) Responsive Speech:

1. எதனால் எழுதலாம்.
2. பாலின் நிறன் என்ன?
3. ஒரு வாரத்திற்கு எத்தனை நாட்கள்?
4. பஸ்ஸை யார் ஓட்டுகிறார்?
5. ஸ்டாம்புகள் எங்கு வாங்கலாம்?

Max. Score: 10
Patient's Score:

SCORE

Language parameters	Maximum Score	Patient's subscores	Total for AQ
I. SPONTANEOUS SPEECH:			
Information content	10		
Fluency	10		
TOTAL	20		
II. COMPREHENSION			
Yes-No questions	60		
Auditory Word Recognition	60		
Sequential Commands	80		
TOTAL	200		
(Divided by 20)	10		
III. REPETITION	100		
TOTAL	100		
Divided by 10	10		
IV. NAMING			
Object Naming	60		
Word Fluency	20		
Sentence Completion	10		
Responsive Speech	10		
	100		
Divided by 10	10		

APHASIA QUOTIENT:

Add and multiply the total by 2

அறிவிக்கப்பட்ட **சம்மதப்படிவம்**

ஆய்வு தலைப்பு.....

ஆய்வு எண்.....

நோயாளியின் முழு பெயர்.....

பிறப்பு / வயது தேதி.....

முகவரி.....

.....

.....

சான்றளிப்பு: நான் சான்றளிக்கிறேன்

1. நான் இங்கு சொல்லப்பட்ட ஆய்வுபற்றிய தகவல் தாளை படித்து முழுமையாக புரிந்து கொண்டேன். எனக்கு கேள்விகள் கேட்பதற்கான வாய்ப்பு வழங்கப்பட்டது
2. மருத்துவர் எனக்கு ஆய்வின் தன்மையை விளக்கினார். எனக்கு கேள்விகள் கேட்பதற்கான வாய்ப்பு வழங்கப்பட்டதுஆய்வில் எனது பங்கேற்பு எனது தனிப்பட்ட முடிவாகும்.
3. எந்த நேரத்திலும் இந்த ஆய்விலிருந்து விலக எனக்கு முழு உரிமை உள்ளது என்று எனக்கு தெரியும். எனது நோய்க்கான மருத்துவம் மற்றும் எனது சட்ட உரிமைகள் பாதிக்கப்படாது என்று புரிகிறது.
4. இந்த மருத்துவ கல்லூரியின் நிறுவன ஒழுக்கவியல் குழு மற்றும் கட்டுப்பாட்டு அதிகாரிகள் தற்போதைய ஆய்வு மற்றும் அது தொடர்பாக நடத்தப்படும் ஆராய்ச்சி மற்றும் பதிவுகளை ஆய்வு செய்திடுவர்கள் என்றும், அதற்கு அனுமதி தேவையில்லை என்றும் புரிகிறது
5. எனது அடையாளத்தை மூன்றாம் நபர்களிடம் தெரியபடுதமட்டர்கள் என்று புரிகிறது. இந்த ஆய்வில், அறிவியல் நோக்கத்திற்காக எழுதபடுகின்ற அராய்ச்சி முடிவுகள் மற்றும் அறிவியல் தகவல்கள் சமுக நோக்கிற்காக பயன்படுத்துவதை ஒப்புகொள்கிறேன்
6. நான் மேலே கண்ட ஆய்வில் பங்கேற்க ஒப்புகொள்கிறேன்

நோயாளியின் / பிரதிநிதி கையொப்பம்(விரல் ரேகை)

கையொப்பமிட்டவர் பெயர் / தேதி

ஆய்வு விசாரணை செய்பவரின் பெயர்

விசாரணை செய்பவரின் தேதி கையொப்பம்

சாட்சி தேதி கையொப்பம்

NIH STROKE SCALE

1.a. Level of Consciousness:	0 Alert
	1 Not alert, but arousable with minimal stimulation
	2 Not alert, requires repeated stimulation to attend
	3 Coma
1.b. Ask patient the month and their age:	0 Answers both correctly
	1 Answers one correctly
	2 Both incorrect
1.c. Ask patient to open and close eyes and	0 Obeys both correctly
	1 Obeys one correctly
	2 Both incorrect
2. Best gaze (only horizontal eye movement):	0 Normal
	1 Partial gaze palsy
	2 Forced deviation
3. Visual Field testing:	0 No visual field loss
	1 Partial hemianopia
	2 Complete hemianopia
	3 Bilateral hemianopia (blind including cortical blindness)
4. Facial Paresis (Ask patient to show	0 Normal symmetrical movement
teeth or raise eyebrows and close eyes	1 Minor paralysis (flattened nasolabial fold, asymmetry on smiling)
tightly):	2 Partial paralysis (total or near total paralysis of lower face)
	3 Complete paralysis of one or both sides (absence of facial movement in the upper and lower face)
5. Motor Function - Arm (right and left):	0 Normal (extends arems 90 (or 45) degrees for 10 seconds without drift)
	1 Drift
Right arm ____	2 Some effort against gravity
Left arm ____	3 No effort against gravity

	4 No movement
	9 Untestable (Joint fused or limb amputated)
6. Motor Function - Leg (right and left):	0 Normal (hold leg 30 degrees position for 5 seconds)
	1 Drift
Right leg ____	2 Some effort against gravity
Left leg ____	3 No effort against gravity
	4 No movement
	9 Untestable (Joint fused or limb amputated)
7. Limb Ataxia:	0 No ataxia
	1 Present in one limb
	2 Present in two limbs
8. Sensory (Use pinprick to test arems, legs,	0 Normal
trunk and face -- compare side to side):	1 Mild to moderate decrease in sensation
	2 Severe to total sensory loss
9. Best Language (describe picture, name items,	0 No aphasia
read sentences)	1 Mild to moderate aphasia
	2 Severe aphasia
	3 Mute
10. Dysarthria (read several words): 0 Normal articulation	
	1 Mild to moderate slurring of words
	2 Near unintelligible or unable to speak
	9 Intubated or other physical barrier
11. Extinction and inattention:	0 Normal
	1 Inattention or extinction to bilateral simultaneous stimulation in one of the sensory modalities
	2 Severe hemi-inattention or hemi-inattention to more than one modality

NIHSS SCORE -

MASTER CHART - I

CASE .NO	AGE	SEX	LITERACY	HANDEDNESS	RISK FACTORS	NIHSS SCORE	ASSOCIATED DEFECTS	INITIAL APHASIA TYPE	EVOLUTION OF APHASIA AT END OF 6 MONTHS	APHASIA QUOTIENT			CT SCAN & SIZE OF THE INFARCT
										1st day	4th week	24th week	
1	77	F	IL	R	DM/HT	20	Right Hemiparesis	Global		2.8	-	-	3X2.5cm Left fronto tempero parietal Infarcts including Broca's, Wernicke' Supra marginal gyrus, Cortical and su cortical regions
2	68	M	L	R	HT	14	"	Wernicke		20.2	-	-	2x1cm Left tempero parietal infarct including Wernicke's, Supra marginal gyrus, Cortical and subcortical region
3	65	M	L	R	-	18	"	Global	Global	2.8	2.8	11.6	2x2.5cm Left fronto tempero parietal Infarcts including Broca's, Wernicke' Supra marginal gyrus, Cortical and su cortical regions
4	57	M	IL	R	DM/HT	22	"	Global	Global	2.8	11.2	24	3x2cm "
5	71	F	IL	R	HT	20	"	Global		2.6	-	-	3x2.5cm "
6	65	F	IL	R	HT/CAD	17	"	Global		8	-	-	3.5x2cm "
7	37	M	L	L	DM/A/S	15	"	Global	Broca	14.4	39.6	66	3x2cm "
8	55	M	L	R	-	14	"	Global	Broca	24	42.2	62.4	2x2.5cm "
9	60	M	L	R	-	23	"	Global	Global	2.8	2.8	4.2	3.5x2cm "
10	65	M	IL	R	-	24	"	Global		2.8	-	-	3x2.5cm "
11	41	F	L	R	RHD	22	"	Broca		27.04	-	-	2x2.5cm Left frontal Infarcts Cortica and subcortical in Broca's and adjace areas
12	45	M	L	R	-	34	"	Global	Global	2.8	2.8	4.2	3x2cm Left fronto tempero parietal Infarcts including Broca's, Wernicke' Supra marginal gyrus, Cortical and su cortical regions

13	33	M	L	R	S/A	15	"	Broca	Transcortical Motor	27.04	50.8	75.2	2x1cm Left frontal Infarcts Cortical a subcortical in Broca's and adjacent Areas
14	50	M	IL	R	-	15	"	Global	Broca	7.2	30.4	51.6	10 – 15ml Left fronto tempero parieta haemorrhage including Broca's, Wernicke's, Supra marginal gyrus, Cortical and subcortical regions
15	65	M	L	R	-	23	"	Global	Global	2.8	3.6	3.6	15 – 20ml "
16	31	M	L	R	A	13	"	Wernicke	Wernicke	22.8	33	42.4	3.5x2cm Left tempero parietal infarct including Wernicke's, Supra marginal gyrus, Cortical and subcortical regions
17	55	F	IL	L	RHD	20	"	Global	Broca	12	35.6	56.4	3x2cm Left fronto tempero parietal Infarcts including Broca's, Wernicke' Supra marginal gyrus, Cortical and su cortical regions
18	32	F	L	R	-	18	"	Global		9.8	-	-	3x2.5cm Left MCA infarct with haemorrhagic transformation
19	45	F	L	R	HT/CAD	28	"	Global	Broca	12	37.4	57.4	2x2.5cm Left fronto tempero parietal Infarcts including Broca's, Wernicke' Supra marginal gyrus, Cortical and su cortical regions
20	50	F	IL	R	HT	32	"	Global		3.6	-	-	3x2.5cm "
21	65	M	IL	R	-	25	Right Hemiparesis	Global		4.8	-	-	3x2.5cm Left fronto tempero parietal Infarcts including Broca's, Wernicke' Supra marginal gyrus, Cortical and su cortical regions
22	28	F	L	R	-	6	No Hemiparesis	Broca	Transcortical Motor	60.4	65.2	89.6	0.5x1cm Left frontal Infarcts Cortica and subcortical in Broca's and adjacer Areas
23	43	M	L	R	HT/CAD	5	Right Hemiparesis	Transcortical Motor		50.8	-	-	2x2.5cm Left frontal Infarcts anterio and superior to Broca's area
24	52	M	IL	R	HT/DM	21	"	Wernicke		22.6	-	-	10 – 15ml Left tempero parietal haemorrhage including Wernicke's,

													Supra marginal gyrus, Cortical and subcortical regions
25	58	M	L	R	HT/CAD	27	"	Wernicke		21.4			10 – 15ml Left tempero parietal haemorrhage including Wernicke's, Supra marginal gyrus, Cortical and subcortical regions
26	70	F	IL	R	-	30	"	Global		2.8			3x2cm Left fronto tempero parietal Infarcts including Broca's, Wernicke' Supra marginal gyrus, Cortical and su cortical regions
27	35	M	L	R	HT/S/A	13	"	Global	Broca	7.2	36	49.6	15 – 20ml Left fronto tempero parieta Haemorrhage including Broca's, Wernicke's, Supra marginal gyrus, Cortical and sub cortical regions
28	42	M	L	R	HT/S	7	"	Broca	Broca	25	34	43.6	1x1cm Left frontal Infarcts Cortical a subcortical in Broca's and adjacent Areas
29	62	M	L	R	DM/HT	21	"	Global	Global	2.8	4.2	16.2	3.5x2cm Left fronto tempero parietal Infarcts including Broca's, Wernicke' Supra marginal gyrus, Cortical and su cortical regions
30	48	M	L	L	-	27	"	Global		3.6			3x2.5cm Left fronto tempero parietal Infarcts with haemorrhagic transformation including Broca's, Wernicke's, Supra marginal gyrus, Cortical and subcortical regions
31	53	M	L	R	-	7	"	Broca		16			2x1cm Left frontal Infarcts Cortical a subcortical in Broca's and adjacent Areas
32	44	M	L	R	S/A	18	"	Global	Broca	13.2	35	57.6	3x2.5cm Left fronto tempero parietal Infarcts including Broca's, Wernicke' Supra marginal gyrus, Cortical and su cortical regions
33	50	M	L	R	-	19	"	Global	Global	2.8	3.6	4.2	15 – 20ml Left fronto tempero parieta

													Haemorrhage including Broca's, Wernicke's, Supra marginal gyrus, Cortical and sub cortical regions
34	63	M	IL	R	HT/CAD	12	"	Global		2.8			3x2cm Left tempero parietal infarct including Wernicke's, Supra marginal gyrus, Cortical and subcortical regions
35	61	F	L	R	-	23	"	Wernicke	Wernicke	18.4	21.6	30	3.5x2cm "
36	47	M	L	R	HT/CAD/S	6	"	Wernicke	Wernicke	20.4	24	28.4	3.5x2cm "
37	75	M	IL	L	-	32	"	Wernicke		16.8			15 – 20ml Left tempero parietal haemorrhage including Wernicke's, Supra marginal gyrus, Cortical and subcortical regions
38	63	F	IL	R	DM	13	"	Global		2.6			3x2cm Left fronto tempero parietal Infarcts including Broca's, Wernicke' Supra marginal gyrus, Cortical and su cortical regions
39	29	M	L	R	RHD	6	"	Broca	Transcortical Motor	58	65.2	89.6	1x1.5cm Left frontal Infarcts Cortica and subcortical in Broca's and adjacer areas
40	61	F	IL	R	HT	24	"	Conduction		81.7			1x1.5cm Left tempero parietal infarct involving sub cortical regions sparing Wernicke's area
41	54	M	IL	R	-	21	Right Hemiparesis	Global	Global	2.0	10.2	22.2	3x2cm Left fronto tempero parietal Infarcts including Broca's, Wernicke' Supra marginal gyrus, Cortical and su cortical regions
42	72	F	L	R	-	19	"	Broca		58			2x1cm Left frontal Infarcts Cortical a subcortical in Broca's and adjacent Areas
43	49	M	L	R	S/A	12	"	Global	Broca	8.2	36	52	2x2.5cm Left fronto tempero parietal Infarcts including Broca's, Wernicke' Supra marginal gyrus, Cortical and su cortical regions

44	75	M	IL	R	HT/DM/S	22	“	Global	Global	2.8	5.2	17.2	3.5x2cm “
45	60	M	L	R	-	14	“	Global	Broca	13.2	48.8	60	3x2.5cm “
46	46	M	IL	R	DM/S/A	13	“	Global	Broca	7.4	30.8	52	3x2.5cm“
47	75	M	L	L	HT/CAD	24	“	Global	Global	3.6	4.2	4.2	15 – 20ml Left fronto tempero parieta Haemorrhage including Broca’s, Wernicke’s, Supra marginal gyrus, Cortical and subcortical regions
48	60	M	L	R	-	28	“	Global	Global	3.6	4.2	4.2	3.5x2cm Left fronto tempero parietal Infarcts including Broca’s, Wernicke’ Supra marginal gyrus, Cortical and su cortical regions
49	28	M	L	R	S/A	5	“	Broca	Transcortical Motor	29.04	54.8	65.2	1x1.5cm Left frontal Infarcts Cortica and subcortical in Broca’s and adjacer Areas
50	63	F	L	R	DM/HT	19	“	Global		4.2			3x2cm Left fronto tempero parietal Infarcts including Broca’s, Wernicke’ Supra marginal gyrus, Cortical and su cortical regions
51	37	M	L	R	-	10	“	Broca	Broca	22	36.4	50.2	1x1cm Left frontal Infarcts with haemorrhagic transformation Cortical and subcortical in Broca’s and adjacent areas
52	80	F	IL	R	DM/HT	21	“	Wernicke		21.8			2x2.5cm Left tempero parietal infarct including Wernicke’s, Supra marginal gyrus, Cortical and subcortical region
53	55	M	L	R	HT/CAD/A	32	“	Global	Global	2.8	8.2	24.2	3x2cm Left fronto tempero parietal Infarcts including Broca’s, Wernicke’ Supra marginal gyrus, Cortical and su cortical regions
54	60	M	IL	R	-	25	“	Wernicke		20.8			15 – 20ml Left tempero parietal haemorrhage including Wernicke’s, Supra marginal gyrus, Cortical and subcortical regions
55	58	M	L	R	-	14	“	Global	Broca	13.2	39	62	3x2.5cm Left fronto tempero parietal

													Infarcts including Broca's, Wernicke' Supra marginal gyrus, Cortical and su cortical regions
56	60	M	L	R	DM	19	"	Broca		50.8			2x1cm Left frontal Infarcts Cortical a subcortical in Broca's and adjacent Areas
57	54	M	IL	L	A	21	"	Wernicke	Wernicke	22	28	30	3x2cm Left tempero parietal infarct including Wernicke's, Supra marginal gyrus, Cortical and subcortical regions
58	68	M	L	R	A/S	23	"	Wernicke		20.8			10 – 15ml Left tempero parietal haemorrhage including Wernicke's, Supra marginal gyrus, Cortical and subcortical regions
59	55	F	L	R	HT	13	"	Wernicke	Wernicke	21.8	30	37.2	3x2cm Left tempero parietal infarct including Wernicke's, Supra marginal gyrus, Cortical and subcortical regions
60	65	M	IL	R	HT	19	"	Global	Global	2.8	12	16.2	3.5x2cm Left fronto tempero parietal Infarcts including Broca's, Wernicke' Supra marginal gyrus, Cortical and su cortical regions
61	65	M	IL	R	HT/CAD	28	Right Hemiparesis	Global	Broca	12	42	65.6	10 - 15ml Left fronto tempero parietal haemorrhage including Broca's, Wernicke's, Supra marginal gyrus, Cortical and subcortical regions
62	62	F	L	R	RHD	21	"	Global		2.8			3x2cm Left fronto tempero parietal Infarcts including Broca's, Wernicke' Supra marginal gyrus, Cortical and su cortical regions
63	57	M	L	R	DM/HT/S	20	"	Global	Global	2.8	11.6	15	15 – 20ml Left tempero parietal haemorrhage including Wernicke's, Supra marginal gyrus, Cortical and su cortical regions

64	55	M	IL	R	-	25	"	Global		4.2			3x2cm Left fronto tempero parietal Infarcts including Broca's, Wernicke' Supra marginal gyrus, Cortical and su cortical regions
65	54	M	L	R	-	19	"	Transcortic al Motor		50.8			2x2.5cm Left frontal Infarcts anterior and superior to Broca's area
66	65	M	IL	R	HT/CAD	18	"	Transcortic al Motor		51.8			1x1cm "
67	58	M	L	L	DM/HT	19	"	Global	Broca	13.4	28.4	54.4	3x2cm Left fronto tempero parietal Infarcts including Broca's, Wernicke' Supra marginal gyrus, Cortical and su cortical regions
68	74	M	L	R	DM/HT/S/ A	21	"	Global	Global	2.8	7.2	9.2	3x2cm "
69	71	M	L	R	-	10	"	Broca	Broca	23	32.4	44.8	0.5x1cm Left frontal Infarcts Cortica and subcortical in Broca's and adjacer Areas
70	58	M	L	R	DM	11	"	Wernicke	Wernicke	25	30.8	32.4	2x2.5cm Left tempero parietal infarct including Wernicke's, Supra marginal gyrus, Cortical and subcortical regions
71	59	M	L	R	-	22	"	Broca		50.8			2x1cm Left frontal Infarcts Cortical a subcortical in Broca's and adjacent Areas
72	41	M	L	R	HT/S	23	"	Global	Global	2.8	9.2	20.2	3.5x2cm Left fronto tempero parietal Infarcts including Broca's, Wernicke' Supra marginal gyrus, Cortical and su cortical regions
73	41	F	L	R	-	13	"	Broca	Broca	21.6	25	34	1x1cm Left frontal Infarcts Cortical a subcortical in Broca's and adjacent Areas
74	70	F	IL	R	HT/CAD	30	"	Global		4.8			15 – 20ml Left tempero parietal haemorrhage including Wernicke's, Supra marginal gyrus, Cortical and

													subcortical regions
75	62	F	L	L	CAD	14	"	Global	Broca	12	41.6	64	3x2.5cm Left fronto tempero parietal Infarcts including Broca's, Wernicke' Supra marginal gyrus, Cortical and su cortical regions
76	48	M	L	R	HT/S	12	"	Global	Broca	7.4	33.6	54	3x2.5cm "
77	42	M	IL	L	HT/S/A	21	"	Global	Global	2.8	9.6	20.2	3x2cm "
78	63	M	L	R	HT/CAD	23	"	Global		4.2			3x2cm Left fronto tempero parietal Infarcts with haemorrhagic transformation including Broca's, Wernicke's, Supra marginal gyrus, Cortical and subcortical regions
79	74	M	L	R	HT/CAD	20	"	Global	Global	2.8	7.2	20.2	3.5x2cm Left fronto tempero parietal Infarcts including Broca's, Wernicke' Supra marginal gyrus, Cortical and su cortical regions
80	52	M	IL	R	HT	19	"	Global	Global	2.8	3.6	5.6	3x2cm "
81	55	M	IL	R	S/A	22	Right Hemiparesis	Global					2x2.5cm "
82	71	F	L	R	DM	24	"	Global					2x2.5cm "
83	68	F	IL	R	DM/HT	13	"	Broca	Broca	22	36.4	51.2	10 – 15ml Left frontal haemorrhage Cortical and sub cortical in Broca's ar adjacent Areas
84	63	M	IL	R	DM	22	"	Wernicke	Wernicke	21.6	28.4	38.8	2x1cm Left tempero parietal infarct including Wernicke's, Supra marginal gyrus, Cortical and subcortical regions
85	58	M	L	R	HT/S/A	19	"	Global	Global	2.8	8.2	22	2x2.5cm Left fronto tempero parietal Infarcts including Broca's, Wernicke' Supra marginal gyrus, Cortical and su cortical regions
86	73	F	IL	R	-	20	"	Broca		22			2x1cm Left frontal Infarcts Cortical subcortical in Broca's and adjacent Areas

87	55	M	L	L	S	13	"	Wernicke	Wernicke	20.8	26	34.8	2x2.5cm Left tempero parietal infarct including Wernicke's, Supra marginal gyrus, Cortical and subcortical regions
88	52	M	L	R	HT/S/A	24	"	Global		2.8			2x2.5cm Left fronto tempero parietal Infarcts including Broca's, Wernicke' Supra marginal gyrus, Cortical and su cortical regions
89	71	M	L	R	HT/CAD	19	"	Wernicke		22			15 – 20ml Left tempero parietal haemorrhage including Wernicke's, Supra marginal gyrus, Cortical and subcortical regions
90	72	M	L	R	-	12	"	Broca	Broca	21	33	49.2	1x1cm Left frontal Infarcts Cortical a subcortical in Broca's and adjacent Areas
91	65	F	IL	R	DM	24	"	Global		2.8			2x2.5cm Left fronto tempero parietal Infarcts including Broca's, Wernicke' Supra marginal gyrus, Cortical and su cortical regions
92	53	F	L	R	HT	6	"	Broca		21			<10 ml Left frontal haemorrhage Cortical and sub cortical in Broca's and adjacent areas
93	51	M	L	R	-	23	"	Global	Global	2.8	2.8	6.8	3x2cmLeft fronto tempero parietal Infarcts including Broca's, Wernicke' Supra marginal gyrus, Cortical and su cortical regions
94	62	M	IL	R	HT/DM	19	"	Global	Global	3.6	2.8	25	15 – 20ml Left fronto tempero parieta haemorrhage including Broca's, Wernicke's, Supra marginal gyrus, Cortical and subcortical regions
95	`53	M	L	R	-	7	"	Wernicke	Wernicke	20.2	29	42.8	2x2.5cm Left tempero parietal infarct including Wernicke's, Supra marginal gyrus, Cortical and subcortical regions

96	52	F	L	R	DM	26	"	Global		4.2			2x2.5cm Left fronto tempero parietal infarct including Broca's, Wernicke's, Supra marginal gyrus, Cortical and subcortical regions
97	57	M	IL	L	HT/DM	21	"	Wernicke	Wernicke	18.4	30.4	32.8	2x1cm Left tempero parietal infarct including Wernicke's, Supra marginal gyrus, Cortical and subcortical regions
98	48	M	L	R	-	25	"	Global	Global	2.8	9.6	20.2	3x2cm Left fronto tempero parietal Infarcts including Broca's, Wernicke' Supra marginal gyrus, Cortical and su cortical regions
99	45	M	L	R	S/A	32	"	Global	Global	2.8	7.6	11.6	3x2cm "
100	49	M	IL	R	HT/CAD	30	"	Global	Global	2.8	2.8	18.6	10 - 15ml Left fronto tempero parietal haemorrhage including Broca's, Wernicke's, Supra marginal gyrus, Cortical and subcortical regions

M-Male L-Literate R-Right Handed HT-Hyper Tension S-Smoking CAD-Coronary Artery Disease

F-Female IL-Illetrate L-Left Handed DM-Diabetes Mellitus A-Alcoholism RHD-Rheumatic Heart Disease

Master Chart II

Global Aphasia Test Score

Pt No	Fluency			Inform Content			Comprehension			Repetition			Naming			Aphasia Quotient		
	T1	T2	T3	T1	T2	T3	T1	T2	T3	T1	T2	T3	T1	T2	T3	T1	T2	T3
3	0	0	0	0	0	0	1.4	1.4	1.8	0	0	4	0	0	0.5	2.8	2.8	11.6
4	0	1	1	0	1	3	1.4	2.1	3.0	0	1	4	0	0.5	1	2.8	11.2	24
7	0	4	6	0	5	8	3.1	4.8	8.0	3.6	4.2	6.0	0.5	1.8	5.0	14.4	39.6	66
8	0	5	5	0	6	8	3.0	4.7	8.2	3.6	4.0	6.0	0	1.4	2.0	24	42.2	62.4
9	0	0	0	0	0	0	1.4	1.4	2.1	0	0	0	0	0	0.5	2.8	2.8	4.2
12	0	0	0	0	0	3	1.4	2.1	3.1	0	0	2	0	0.5	2	2.8	4.2	16.2
14	0	2	3	0	4	7	3.0	4.8	8.0	0.6	3.0	6.0	0	1.4	1.8	7.2	30.4	51.6
15	0	0	0	0	0	0	1.4	1.8	1.8	0	0	0	0	0	0	2.8	3.6	3.6
17	0	2	4	0	5	8	3.0	4.8	8.2	3.0	4.0	6.0	0	2.0	2.0	12	35.6	56.4
19	0	4	5	0	4	8	3.0	4.8	7.8	3.0	4.0	6.0	0	2.0	2.0	12	37.4	57.6
27	0	5	5	0	4	7	3.0	4.0	7.8	0.6	3.0	6.0	0	2.0	2.0	7.2	36	55.6
29	0	00	0	0	0	3	1.4	2.1	3.1	0	0	2	0	0.5	2	2.8	4.2	16.2
32	0	2	3	0	5	7	3.1	4.1	7.8	3.6	4.0	6.0	0	1.8	5.0	13.2	35	57.6
33	0	0	0	0	0	0	1.4	1.8	2.1	0	0	0	0	0	0	2.8	3.6	4.2
41	0	1	1	0	1	2	1.0	2.1	3.1	0	1	3	0	0.5	2	2.0	10.2	22.2
43	0	4	4	0	4	7	3.0	4.0	5.0	0.6	4.0	5.0	0.5	2.0	5.0	8.2	36	52
44	0	0	1	0	0	2	1.4	2.1	3.1	0	0	3	0	0.5	0.5	2.8	5.2	17.2
45	0	5	5	0	6	7	3.0	4.8	8.0	3.6	4.0	5.0	0	1.4	5.0	13.2	48.8	60
46	0	2	3	0	5	8	3.1	4.0	5.0	0.6	3.0	6.0	0	1.4	4.0	7.4	30.8	52
47	0	0	0	0	0	0	1.8	2.1	2.1	0	0	0	0	0	0	3.6	4.2	4.2

48	0	1	1	0	1	2	1.0	2.1	3.1	0	1	3	0	1	2	2.0	12.2	22.2
53	0	0	1	0	0	2	1.4	2.1	3.1	0	1	4	0	1	2	2.8	8.2	24.2
55	0	4	5	0	5	8	3.0	4.7	8	3.6	4.0	5.0	0	1.8	5.0	13.2	39	62
60	0	1	1	0	1	2	1.4	2.0	3.1	0	1	1	0	1	1	2.8	12	16.2
61	0	5	5	0	6	8	3.0	4.0	7.8	3.0	4.0	6.0	0	2.0	6.0	12	42	65.6
63	0	0	0	0	0	0	1.4	1.8	3.0	0	4	4	0	0	0.5	2.8	11.6	15.0
67	0	2	3	0	4	7	3.1	4.1	8.2	3.6	4.0	6.0	0	1.4	2.0	13.4	28.4	54.4
68	0	0	0	0	0	0	1.4	2.1	2.1	0	1	2	0	0.5	0.5	2.8	7.2	9.2
72	0	1	1	0	1	2	1.4	2.1	3.1	0	0	3	0	0.5	1	2.8	9.2	20.2
75	0	5	5	0	6	7	3.0	4.8	8.0	3.0	3.0	6.0	0	2.0	6.0	12	41.6	64
76	0	2	4	0	5	8	3.1	4.0	5.0	0.6	4.0	5.0	0	1.8	5.0	7.4	33.6	54
77	0	1	1	0	1	2	1.4	1.8	2.1	0	1	2	0	1	3	2.8	9.6	20.2
79	0	0	1	0	0	2	1.4	2.1	3.1	0	1	3	0	0.5	2	2.8	7.8	20.2
80	0	0	0	0	0	0	1.4	1.8	1.8	0	0	1	0	0	0	2.8	3.6	5.6
85	0	0	1	0	1	2	1.4	2.1	2.1	0	1	3	0	1	3	2.8	8.2	22.2
93	0	0	0	0	0	0	1.4	1.4	1.4	0	0	1	0	0	1	2.8	2.8	6.8
98	0	1	1	0	1	2	1.4	1.8	2.1	0	1	2	0	1	3	2.8	7.6	11.6
99	0	0	0	0	0	0	1.4	1.8	1.8	0	1	2	0	1	2	2.8	7.6	11.6
100	0	0	0	0	0	3	1.4	1.4	1.8	0	0	4	0	0	0.5	2.8	2.8	18.6

Wernicke's Aphasia Test Scores

Pt No	Fluency			Information Content			Comprehension			Repetition			Naming			Aphasia Quotient		
	T1	T2	T3	T1	T2	T3	T1	T2	T3	T1	T2	T3	T1	T2	T3	T1	T2	T3
16	7	7	8	1	3	3	.5	1.5	1.5	2.6	3.8	4.0	0.3	1.2	1.2	22.8	33	42.4
35	7	7	8	0	1	3	1.4	2.0	3.0	0.8	0.8	1.0	0	0	0	18.4	21.6	30.0
36	7	7	7	0	1	3	1.8	2.0	2.2	1.4	2.0	2.0	0	0	0.9	20.4	24	28.4
57	7	8	8	0	0	1	1.6	2.0	2.0	2.4	3.0	3.0	0	1	1	22	28	30.0
59	6	7	8	0	1	3	1.8	2.0	2.2	2.8	3.8	4.0	0.3	1.2	1.4	21.8	30	37.2
70	7	7	7	1	3	3	2.1	2.4	3.0	1.4	2.0	2.0	0	1	1.2	25	30.8	32.4
84	7	7	8	0	1	3	1.4	2.0	2.2	2.4	3.0	3.0	0	1.2	1.2	21.4	28.4	38.8
87	8	8	8	0	1	3	1.6	2.0	2.2	.8	0.8	1.0	0	1.2	1.2	20.8	26	34.8
95	7	7	8	0	1	3	0.5	1.5	5	2.6	3.8	4.0	0	1.2	1.4	20.2	29	42.8
97	7	7	8	0	3	3	1.4	2.0	2.2	0.8	2.0	2.0	0	1.2	1.2	18.4	30.4	32.8

Broca's Aphasia Test Score

Pt No	Fluency			Inform Content			Comprehension			Repetition			Naming			Aphasia Quotient		
	T1	T2	T3	T1	T2	T3	T1	T2	T3	T1	T2	T3	T1	T2	T3	T1	T2	T3
13	4	4	8	3	4	8	1.72	8	10	1.05	4.4	6.0	4.2	4.4	5.6	27.94	50.8	75.2
22	4	4	9	5	6	9	9	10	10	7	8.0	8.0	4.4	4.6	8.8	60.4	65.2	89.6
28	1	3	4	1	2	4	7.5	8	8.5	1.5	1.5	1.7	1.5	2.5	3.6	25	34	43.6
39	4	4	9	5	6	9	9	10	10	6	8.0	8.0	4.2	4.6	8.8	58	65.2	89.6
49	4	4	9	3	4	4	1.72	8	8	0.6	3.0	6.0	4.2	4.4	8.6	29.04	54.8	65.2
51	1	3	4	1	3	4	7.5	8.0	8.5	1	3.0	5.0	0.5	1.2	3.6	22	36.4	50.2
69	1	3	4	1	2	4	8.0	8.0	8.8	1	2.0	2.0	0.5	1.2	3.6	23	32.4	44.8
73	1	1	2	1	2	4	0	8.5	8.5	0.8	1.0	2.0	0	0	0.5	21.6	25	34
83	1	3	4	1	3	4	7.5	8.0	8.0	1	3	6	0.5	1.2	3.6	22	36.4	51.2
90	1	2	4	1	3	4	6	8	8.0	1	3	5	1.5	2.5	3.6	21	33	49.2